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NEUROLOGICAL INDICATIONS FOR AND AGAINST OPERATION IN TRAUMATIC INJURIES AFFECTING THE CENTRAL NERVOUS SYSTEM.

BY JOHN JENKS THOMAS, M.D., BOSTON.

IN the time allowed for the presentation of one paper in a symposium on the surgical treatment of injuries of the brain and spinal cord, one can touch only upon some of the more important points, and I beg your indulgence if the statements made in the course of these remarks seem dogmatic. I shall try simply to give briefly the more important conclusions which are the outcome of the observation and study of a considerable number of cases of injury of this nature.

Let us consider first the cases of injury of the spinal column, as these cases present fewer debatable points than those in which there is an injury of the brain.

The spinal cord is damaged in various ways when there is trauma applied to the spinal column, and, in addition, we may have injuries of the cauda or of nerve roots. In falls where the force is applied in the axis of the spinal column, either by striking upon the head, the feet or the buttocks, we may find a compression fracture of the body of one or more vertebrae. In such cases we frequently find no paralysis from injury of the nerve structure at the point of fracture though, in recent cases, a band of hyperalgesia on the trunk or alterations of the epigastric or abdominal reflexes may indicate injury of the thoracic nerve roots as they pass through the intravertebral foramina. Aside from these signs on the part of the nervous system, such fractures can frequently be diagnosed from radiograms. Such cases need to be differentiated from sprains of the back, by the use of radiograms, and are best treated by fixation of the injured portion of the spine by plaster of Paris or apparatus.

In addition to injuries of the nerve roots, these cases not infrequently show areas of hemorrhage or softening in the substance of the spinal cord, at times far removed from the seat of the fracture. Experience in the present war has shown that similar areas of damage to the spinal cord are frequently produced by the impact of high velocity missiles upon the bones of the spine, not only the bodies of the vertebrae, but also the transverse and other processes.

Such a case was that of Eugene C., seen by me in consultation on Jan. 18, 1914. The young man, 25 years of age, about one year before, fell a distance of 30 feet, landing on the buttocks, and showed immediate pricking sensation and pain in the legs, complete paralysis of both legs and retention of urine, followed after two months by difficulty in retaining the urine. There had been gradual partial improvement, but he still showed marked paralysis with atrophy in the buttocks and anterior tibial groups of muscles. There was loss of skin sensation over the buttocks, backs of the thighs and

in the legs below the knees, with good sensation in the front of the thighs to a point a little below the knees. The knee jerks were diminished, there was a double ankle clonus, but no Babinski or Oppenheim's signs. There was a rounded kyphosis in the lower thoracic and upper lumbar regions, with fixation, and an x-ray showed a crush of the 1st lumbar vertebra. This case was much benefited by tenotomies of the contracted tendo Achillis and apparatus.

Quite frequently the area of destruction in the cord is found to be unilateral, when there is usually present at first the Brown-Séquard syndrome of loss of superficial sensation in the opposite side of the body from the lesion, with loss of motion and deep sensation on the same side.

Such a case was that of H. A., who was seen by me on April 21, 1917. He was struck by a motor car, knocked down and showed no voluntary motion in the right arm and hand and moderate paresis of the right leg. Strength in the left extremities was good. Skin sensation was much diminished on the left side of the trunk, extending as high as the first rib, and moderately diminished in the left leg. The sense of position was much disturbed in the right hand and arm and slightly in the right leg. Bony sensation as tested by vibration was practically equal on the two sides. The triceps, biceps and radial reflexes on the right were absent, the right knee jerk was increased and there was slight ankle clonus on that side with Babinski's sign, but normal reflexes on the left side. There was marked tenderness of the neck and the x-ray plate showed a slight displacement of the second cervical vertebra.

Cases of this nature, as shown by the results of treatment and the conditions found at necropsy, are not benefited by immediate surgical operation. The treatment should be limited to fixation of the injured vertebral column, in order to promote rapid healing of the injury, and also to prevent further injury of the cord, in case there has been rupture of the ligaments so extensive that displacement of the bones of the spine may occur from handling the patient. This danger is greatest in injuries of the cervical spine.

Cases of dislocation, or fracture-dislocation of the spine in all regions without signs of injury of the cord, require simply fixation of the spine.

More debatable is the treatment of the cases of fracture-dislocation of the spine with injury of the cord. These injuries of the cord are produced almost invariably by displacement forward of the upper fragment of the injured spine, the cord being crushed between the lowest arch of the displaced fragment and the body of the vertebra below. The weight of evidence from x-ray examinations and examinations of these fractured spines at necropsy shows very conclusively that this forward displacement can almost invariably be overcome entirely by fixation of the spinal column in hyperextension. The difficulties arise in maintaining this. Rarely cutting down upon the spine and wiring is required. Hyperextension and fixation is the best treatment of all cases where the examination of the functions of the nervous system shows we have to do with a partial destruction of the cord, as where sensation either for the deeper structures or for the skin is partially retained. The immediate loss of all sensation in joints, knees, muscles and skin, the complete loss of voluntary motion, with absence of the knee and ankle jerks, and loss of control of the bladder, when all present, are in favor of a total transverse lesion of the cord. In addition, recent observations seem to show a special importance attaching to the plantar reflex in the estimation of the amount of damage done to the cord. In milder cases with loss of motion and sensation, the plantar reflex is of the extensor type, often with flexion of the thigh, and dorsi-flexion of the foot—Babinski's sign. In severer cases there is found, on testing for the plantar reflex, occasional flexion of the toes, or a total loss of any response.

It is useless to operate in cases of total crush of the spinal cord, unless it is found to be impossible to prevent slipping of the fragments of the fractured spine by any method of fixation. In cases of partial injury of the cord, immediate operation of laminectomy, or this procedure within a few days, is of no advantage. Cases of injury of the cord which are not operated upon early and show improvement, with retrogression of power later, should be again studied by radiographic pictures to determine the possible compression of the spinal cord by callus. In quite a different class stand another group of cases in which the vertebral arches have been driven down upon the cord, compressing it by fragments of bone. Such cases demand immediate operation just as much as do depressed fractures of the skull. This condition is extremely rare except in cases where the patient has struck the back against some small object during the fall, such as an awning stanchion, or in cases of gunshot wounds of the back. In both cases a positive diagnosis of compression by fragments of bone may be obtained by means of good x-ray plates. Crepitus is of little importance in this connection as it is most often found where there is fracture of the spinous process only and is most frequently absent with fracture of the arch.

I may speak briefly of one other condition in which laminectomy should be considered. This is in cases where the neurological examination indicates a complete crushing of the cord, especially in the cervical region, where the outlook is practically hopeless, and where the patient is seen immediately after the receipt of the injury. In these extreme cases, if the operation can be done within two or three hours, and certainly not later than six hours, after the receipt of the injury, a laminectomy, with careful longitudinal hemisection of the cord, may be

tried in the hope of relieving pressure upon nerve structures by the release of hemorrhage within the substance of the cord. In such desperate cases one must not be disappointed by the general absence of any benefit from the operation, as the cases in which this can be expected are few. The only case I have seen early enough after the injury to advise this method of procedure died in about the average length of time after the fall, with no evidence of any benefit; but I may add, the operation, so far as we could judge, did nothing to hasten the end.

I can only say, while, like most aphorisms, it goes too far, that I agree with the witty writer who stated that surgeons no longer operate for fracture of the spine—only some Americans do it.

The question of indications for and against operation in fractures of the skull is, if anything, more complicated than that for injuries of the spinal cord.

In certain forms of injury of the brain this matter can be stated very briefly in its broader aspects. First, in regard to the depressed fractures of the vault of the skull: Where these are compound, the condition of the bone should always be carefully examined, and loose fragments removed or elevated. Fractures of this form where the scalp is uninjured, if slight, and if an x-ray shows no distinct depression of the inner table of the skull, should usually be left undisturbed, but in cases where there is distinct depression or where there is some focal sign of injury of the brain beneath, such as a cerebral monoplegia, astereognosis or aphasia, should be operated upon, and enough of the bone removed to enable one to judge of the amount of damage done to the brain beneath, though, as a rule, little should be attempted beyond the removal of blood clot, so far as it is practicable, and the relief of intracranial pressure by opening the dura if this is high.

In the second place, let us consider the conditions in the bursting fractures of the skull. These have been shown by many writers to be in the nature of fissures, often multiple, which run to the base of the skull. When not compound, either externally or in the ear, or nasal and pharyngeal cavities, with the attendant danger of infection and the development of meningitis or cerebral abscess, these cases require no operative interference except to relieve damage to the cranial contents. The well-known cases of compression of the brain by hemorrhage from the middle meningeal artery, always require operation to remove the clot and check the hemorrhage. In these cases and also in other fissures of the base, a great deal of information may be obtained through lumbar puncture. By this means we can measure with a manometer the pressure of the cerebrospinal fluid in the spinal canal, and, by withdrawing fluid, give some temporary relief if this is increased, and, in addition to this, the finding of macroscopic blood in the fluid, especially if this remains the same in the last fluid drawn as in the first, is of the greatest use. The classical signs of hemorrhage from the middle meningeal artery are well known. The passing away of the initial confusion or unconsciousness with a clearer period followed by gradually increasing unconsciousness, while not diagnostic, as they may be seen in other forms of cerebral trauma, are important, especially when accompanied by convulsive or twitching movements of one side of the body, or paresis which is partial and more marked in the face and arm than in the leg, showing the greatest pressure at the base of the skull over the cortical areas for the facial muscles which lie nearest the point of rupture of the artery. The increase of tendon reflexes on this side, the presence of Babinski's extension of the great toe in testing for the plantar reflex, and the loss of the abdominal reflexes on the same side, are of more importance in deciding that we have compression of one side of the brain than the severity of the paralysis. Usually inequality of the pupils, varying according to the amount of compression of the brain, and conjugate deviation of the eyes may also be used as evidence of unilateral injury of the brain.

A. K. D., age 20, was seen by me in consultation on Oct. 20, 1914. He had been struck on the head at about 2.30 p. m. of that day and was dazed simply, but went home after lying down a short time. At 5.30 p. m., he was found to be very dull, but could be made to walk, and at 6 p. m. he could not be roused. At that time, he showed the slow pulse of cerebral compression, it being 48, to the minute, and did not move the right extremities. He had vomited a couple of times, once during the examination. He was very dull, but protruded the tongue when told, but did not answer questions. There was no voluntary motion of the right arm seen, but the leg was moved at times. All tendon reflexes on the right were increased, with ankle clonus, Babinski, and Oppenheim's signs on the right, but not on the left. The cremasteric reflex was lost on the right and the abdominals much diminished. There was paresis also of the right side of the face. The pupils were of medium size, equal, and reacted moderately well to light. There was some slow oscillation of the eyes, but no conjugate deviation. The blood pressure was 120. Operation was done at once and blood clot estimated at 6 to 8 oz. removed, and the middle meningeal artery, which was bleeding, was tied. The clot was extra-dural. Recovery was complete and uneventful.

In general, in cases of fissure of the skull, we do not have to deal with a gradually increasing intracranial hemorrhage which destroys life by the increase of intracranial pressure, though this condition, whether due to rupture of the meningeal artery or of a basal or other sinus, demands operation for its relief. Hemorrhage from a sinus is much more difficult to reach and check, but, on the other hand, as the pressure in sinuses is less than in arteries, it can more often be controlled by packing, if located in situations where this can be used.

In the great majority of cases of fissure of

the base of the skull, however, the damage to the brain consists in the traumatic edema which we know, from the work of many experimenters, follows after blows upon the head, or transmitted to it through the spine, or in lacerations of brain substance with more or less consequent destruction of brain tissue—or a combination of these two. Before considering in any case the advisability of operation, we must have a clear conception as to what can be accomplished by operation. It is obvious that excision of damaged brain tissue producing focal symptoms, cannot relieve them, and only adds to the cerebral trauma. The one thing which can be done by operation in injuries of the cerebral tissues themselves is the relief of intracranial pressure. This can be done for a short period by lumbar puncture, but as the cerebrospinal fluid is formed very rapidly (Frazier estimating that it is replaced on the average every six hours) this procedure must necessarily be repeated in, at most, that length of time. The so-called decompression operation, of removing a portion of bone and opening the dura, offers a much more effective method of relieving the intracranial pressure for longer periods of time. Obviously, such a measure cannot save all cases, and if widespread lacerations of the brain are present, it does very little even to prolong life. What now are the chief symptoms of increased intracranial pressure? They are loss of consciousness, headache, slowing of the pulse rate, blurring of the optic discs, a steadily rising blood pressure, especially if the rise be fairly rapid, and severe vomiting, or occasionally convulsions. Inequality of the pupils, deviation of the eyes, paralysis, local twitching, unilateral changes in reflexes belong among the signs by which, if they are not changeable, we may localize the point, or at least the hemisphere, of greatest injury or edema. I venture to make the statement that in fracture of the skull, signs of local injury of the brain are never, in themselves, an indication for operation. The next question and the one most difficult of all to decide is whether the symptoms in a given case are due to increased intracranial pressure, with moderate cerebral lacerations, or whether these latter predominate and render operation for relief of pressure useless.

In considering the first question, whether intracranial pressure is so much increased in a certain case as to require relief by operation, the general signs, as distinguished from focal signs, are much the more important. Of these, deepening unconsciousness, slowing of the pulse, blurring of the outlines of the optic discs and a steadily rising blood pressure—or much better, in cases where this can be carried out, an increasing pressure of the cerebrospinal fluid, as measured by a manometer, by means of lumbar puncture—are the most important. Let me emphasize here that in speaking of the blood pressure, it need not by any means be high, the important point being that it is rising—not the absolute pressure. We know that cases which otherwise would pretty certainly prove fatal, can be saved by an operation to lessen the intracranial pressure, and in almost all the cases the period of convalescence is shortened, and the irritability, headache and other symptoms generally seen after severe cerebral concussions, are much lessened.

On the other hand, widespread laceration of brain tissue, from hemorrhage into the cerebral substance, is influenced but little in its course or outcome by the relief of the increased intracranial pressure which is generally found in these cases. Have we, then, any means of knowing how extensive damage has been done by these lacerations? Phelps has shown that these are usually quite widespread, and their most common seat is at the base of the brain in the frontal lobes and at the tips of the temporal lobes. Aside from the fact that extensive destruction of cerebral tissue at these points produces no symptoms of loss or disturbance of function by which it can be recognized, the unconscious or confused mental state of these patients precludes any examination of such brain functions as taste, smell, or speech. For these reasons, again, we have to depend upon objective signs which may not always be reliable to indicate to us that the cerebral tissue damage has been from actual destruction rather than from pressure. The most reliable signs of this sort, the presence of which means that an operation offers no probable relief to the conditions present, are these: First, I should place fixed, rigid pupils, most often dilated, which show no reaction to light; and in the second place, steadily rising temperature which, in the absence of infection, is one of the most reliable signs of extensive destruction of cerebral tissue we have. In these cases, in spite of the fact that intracranial pressure can be relieved, the final outcome remains uninfluenced. I need hardly add to these contraindications for operation in injuries of the brain, that the presence of profound shock is always a contraindication to operation.

Let me mention another pitfall into which one may stumble rather easily in deciding for or against operation in injuries of the head. These are cases of apoplexy or intracerebral hemorrhage, which, in falling, have received an injury of the head. I may instance one such case seen recently at the Boston City Hospital, where a woman of about fifty years of age, who was going down a flight of stairs, swayed and fell headlong, striking the head. On entrance, she had a moderately severe scalp wound exposing the bone, where a fissure could be felt by the probe. The blood pressure was moderately high and bloody fluid, drawn in three separate tubes, was obtained by lumbar puncture. The patient was in deep stupor, but moved the extremities of one side and responded to pin pricks, and showed a distinct flaccid paralysis of the face,

arm and leg of the other side, with ankle clonus. Babinski's sign of slow extension of the great toe, on testing for the plantar reflex on that side was present, with loss of the abdominal and epigastric reflexes on the same side. Here the fact that the paralysis was more complete than is seen in cases of cerebral laceration, who show similar responses to stimulation; then, too, the fact that the paralysis was approximately uniform in face, arm, and leg, which is rarely seen in cortical injuries, and the loss of the skin reflexes on the paralyzed side, led us to decide in favor of cerebral hemorrhage complicated by fracture of the base. The rapid clearing up of the disturbance of consciousness, and the persistence of the marked hemiplegia, amply confirmed this conclusion.

In closing, I wish to mention one further class of cases of fracture of the skull in which operation is of value. In certain cases where we have found perhaps no evidence of paralysis and no definite reasons for operation, either exploratory or decompressive, the patient may improve up to a certain point, then show a persistent mental slowness, perhaps with irritability. In a few such cases in which I have advised opening the cranial cavity there has been found a dura with an organizing blood clot adherent, and marked and rapid improvement in the mental condition has followed almost immediately the removal of this clot, or the lessening of tension by opening the dura, when the clot was not accessible. In cases where these results were seen, there was always found at operation a marked diminution of the cerebral pulsation as compared with that found in normal brains.

Finally, let me state that in deciding in favor of operation in fracture of the skull, we should always have in mind that the most important object aimed at is the relief of increased intracranial pressure, whether this be due to extracerebral hemorrhage or to cerebral edema, and one should strive to determine in each individual case whether the relief of this pressure is likely so to relieve the threatening condition as to warrant the added risks of cranial operation.

DISCUSSION.

DR. P. C. KNAPP, Boston: I am more than disposed to agree with the conclusions which Dr. Thomas has made concerning the operations for fractured spine. Theoretically, of course, or imaginatively, we may have a hemorrhage proceeding slowly which might possibly compress the spinal cord. I doubt this very much, but it can be imagined, and theoretically, too, we may have a fracture of the spine which goes on so slowly and gradually, as when the arches are crushed in, that the fragments of bone may come down gently on the spinal cord, and compress it and cut off its function. I do not believe such cases exist very often. I certainly have been looking for them for many years and have not found them. The damage to the spinal cord is done when the spine is broken. The sudden fracture of the spine or the driving in of the arches smashes the cord. The harm is done and no operation can benefit it.

The rules of diagnosis of a complete transverse lesion of the cord, which, of course, would preclude any operation, are more or less familiar, but at the same time they are not wholly to be depended upon. Dr. Thomas has spoken of the absence of the plantar reflexes as being indicative of a complete transverse lesion of the cord. I saw one case where laminectomy was done and the operator looked down and saw the two ends of the cord divided, and the boy had a year afterwards a persistent plantar reflex. Then, again, you may get a pretty complete destruction of the spinal cord when sensation is retained. I remember one case where the patient had almost no anesthesia, with just the slightest difference in the sensation above and below the seat of the lesion, yet the autopsy showed a transverse band of softening straight across the cord, and microscopic examination failed to show any normal healthy fibre in that portion of the cord.

Ever since Dr. Alfred Allen of Philadelphia brought forward the project of longitudinal section of the cord in cases of acute softening after injury, I have been hoping to see such a case in time. I never yet have seen one soon enough and am skeptical about the benefit from such a procedure. The theory depends entirely upon experimental evidence, and has not been confirmed to any alarming extent by operative experience.

On the whole, therefore, I feel that any operation upon the spinal cord in case of injury or of spinal fracture or dislocation is of very questionable expediency. The cases that seemed most hopeful in my own experience have proved most disappointing. Once in a great while I suggest operation, but am generally sorry.

In regard to fractures of the skull, it may seem somewhat paradoxical, but a fracture of the skull is a comparatively trifling injury. The fracture *per se* is nothing, except as the brain is injured beneath, and the thing which happens to the brain is of course the hemorrhage or laceration. I do not see that it makes the slightest difference whether the bleeding is from the middle meningeal artery, from a small vein going into the longitudinal sinus, or from any other vessel. Now in the injuries to the brain itself, we cannot bank on much information furnished us by our knowledge of brain localization. The whole group of symptoms on the sensory side, including the aphasias and the visual and auditory disturbances, leaves us pretty completely in the lurch, inasmuch as most of the patients are unconscious and we cannot get information of any value concerning them about sensory changes. There are a good many cases of fractured skull that I am disposed to class as x-ray fractures. The patient comes in with a history of injury. For a time he is unconscious, and then comes to, complaining of headache and general uncomfortable conditions without much paralysis, the general condition is fair and the x-ray discloses a beautiful fracture somewhere or other. Some 25 years ago Sir Victor Horsley argued that all cases of fracture should be operated on, inasmuch as there was a chance that epilepsy might develop. I think that is a visionary idea. Later experience has shown comparatively few cases that have developed epilepsy and comparatively few epileptics turn up where we can discover any history or evidence of fracture. To be sure, if the x-ray shows fracture

with displacement of the inner table of the skull, or a fragment driven in anywhere, it would be advisable to operate; but otherwise those cases should be let alone.

Beyond that there are two pathological conditions which arise, the hemorrhage and the oedema and laceration of the brain substance. The only thing we can depend upon in the case of hemorrhage is the existence of paralysis of some sort. If a patient comes in with paralysis, a steadily increasing paralysis, or the simple change in the conditions as shown by a Babinski reflex, or Oppenheim reflex, or difference in the knee jerks, it is wiser certainly to operate.

The abdominal reflexes I consider of comparatively slight value, but in all cases where there is a profound coma and any evidence whatsoever of a paralysis, either evidence we obtain by the patient's inability to move, or by a difference in reflexes, an operation is justificable because that is the one pathological condition we can relieve, and we may and often do save life under these conditions; but the cases of fracture of the base attended by considerable coma, probably increasing, pretty bad general condition, without definite evidence of paralysis more than what is shown by inequality of the pupils or paralysis of some cranial nerve, and especially if there be a good deal of motor restlessness and partial coma, are more strongly suggestive of the laceration which Dr. Thomas has spoken of. Operation may not do very much, but some of the cases certainly are improved by decompression, allowing the brain to drain and any oedema to be relieved.

In such cases we are justified fully in operating for purposes of decompression and that is the one instance where Cushing's subtemporal decompression is of any value. It is certainly of no value in tumors.

Indications for Operation on Traumatic Head Cases.

By Edward H. Nichols, M.D., Boston.

The lesions produced by trauma of the head are numerous and include:

1. Oedema of the brain due to vaso-motor disturbances.
2. Slight laceration of the brain, practically always accompanied by oedema.
3. Local extra-cerebral hemorrhage, either subdural or extra-dural, generally frontal or temporal, and often slight in amount, also accompanied by oedema.
4. Local extra-cerebral hemorrhage of great amount, subdural or extra-dural, generally due to rupture of the middle meningeal artery, also most often temporal or parietal, again accompanied by oedema, sometimes with, sometimes without fracture of the skull.
5. Diffuse extra-cerebral hemorrhage, usually at the base of the brain, generally due to rupture of one of the cerebral sinuses, usually associated with fracture of the base of the skull, and frequently accompanied by a resulting pressure upon or division of one or more of the cerebral nerves.
6. Severe intra-cerebral hemorrhage, due to rupture of cerebral vessels, practically always associated with extensive fracture of the skull.
7. Fractures of the skull which vary from a mere fissure, often detected only by x-ray, or by local swelling, or tenderness, to depressed fracture, sometimes only of the outer table, often involving the entire thickness of the skull, not infrequently compounded; and, finally, fractures of the base of the skull. These latter fractures are usually associated with rupture of one of the cerebral sinuses. These latter fractures are usually produced by compression. A sphere, if compressed, splits along a great circle between the poles of force, but since the skull is not a true sphere, but is buttressed, with numerous openings, the line along which the fracture may occur is very variable. In these cases, as mentioned under No. 5, various injuries of the cerebral nerves may occur, most often the 6th (abducens) and 7th (facial).

Before speaking of the symptoms which may occur with these different classes of injury, it may be well to emphasize the fact that fractures of the skull, *per se*, are of comparatively little importance, except in cases of compound fracture, and the chief danger of these fractures is the possibility of secondary infection of the cerebral meninges, which may occur. The factor of chief importance is the character and extent of the injury to the cerebral tissue, and it is this question, together with the decision as to what must or can be done, that is of chief interest to the surgeon in traumatic head cases. In other words, the fracture usually is of secondary importance; the brain injury is the serious factor.

Consider the symptoms which accompany the different lesions:

1. Oedema of the brain, if uncomplicated, produces symptoms best described under the clinical term *concussion*. These symptoms vary from slight mental confusion to temporary, sometimes prolonged, unconsciousness, or symptoms of cerebral irritation, delirium, and excitability. Such symptoms generally are produced by a blow on the head of moderate severity, a blow, a fall, football collisions, etc. As a result, it is claimed that vaso-motor disturbance of the brain vessels is produced, serum escapes from unruptured vessels, and as the result there is an extra amount of fluid in the brain, and since the skull is unyielding, pressure on the brain occurs. The mental symptoms are variable, depending upon the degree of oedema. There may be a very temporary mental confusion, the patient may talk rationally on simple subjects, but there nearly always is some difficulty in orienting himself,—he cannot tell where he is, the day of the week or month, etc. There practically always is some loss of memory of preceding events, *e. g.*, the patient cannot remember what he had to eat at the preceding meal, or, in severe cases, cannot recall ordinary events that occurred during the twenty-four

hours preceding the injury, and often cannot remember events for a shorter time succeeding the injury. A notable fact regarding the loss of memory is that the so-to-speak "lost time" gradually becomes less as the patient recovers, but a *complete* restoration of memory seldom occurs, even in mild cases. The "automatic" memory often is fairly good, even though actual memory is quite gone. This is shown often in football games, when a man receiving a slight blow may automatically run through play in response to signals fairly accurately, although quite unable to orient himself in response to questions. Or the oedema may produce cerebral irritation, manifested by excitability, or delirium, and the patient in this condition may become violent and assault bystanders, and occasionally such cases are dangerous. If the oedema is severe, unconsciousness may result, partial or complete, but these cases always can be roused, at least partially, by supra-orbital pressure. Vomiting is frequent. The pupils are equal and react, often violently, and usually are dilated; the face is pale, the patient restless and irritable. The *temperature is low*, normal or subnormal, and the pulse rapid. The blood pressure is nearly always low. The spinal fluid is clear and contains no blood. The unconsciousness seldom lasts over twenty-four hours, although some confusion may last longer, and in many of these cases neurasthenic symptoms may last for a considerable time and many of these patients cannot study or do work requiring close mental application for many months.

Treatment. These cases do not need operative treatment. They should be in bed, be given a strong saline cathartic, liquid diet only and an ice cap to the head. The most essential thing is to have such cases carefully watched for twenty-four hours for the occurrence of coma, increasing unconsciousness, spasm, convulsions or paralysis. This is essential, as some cases, showing at first merely symptoms of oedema, later develop intra-cranial hemorrhage and pressure, and no one, at first, can be sure this will not happen.

2. Laceration of the brain. This may occur from direct violence or more commonly from *contre-coup*. In cases uncomplicated by fracture, this laceration generally is slight, and the hemorrhage generally is not great and usually does not produce focal symptoms. The symptoms are practically identical with those of uncomplicated concussion except for two important facts. In cases of laceration, blood will be found in the spinal fluid, and the temperature is elevated (101°-102°). In some of these cases the blood pressure is a little elevated. What has been said about concussion cases applies to the symptoms, prognosis and treatment of these cases of laceration, except that the convalescence is apt to be longer. Occasionally the increased pressure persists and the mental condition remains confused, and rarely these cases should have a subtemporal decompression done, to prevent mental deterioration.

3. Extra-cerebral hemorrhage (intra-cranial hemorrhage). These cases are associated with oedema, the symptom of which they present, accompanied by focal symptoms dependent upon the amount, location and rate of onset of the hemorrhage. They may be divided into three clinical classes—slight (a), severe hemorrhage with focal symptoms (b), and severe without focal symptoms (c).

(a) The slight cases are produced by the rupture of small vessels, generally from a direct blow, *e. g.*, being hit by a pitched ball. The symptoms are those of oedema together with slight focal paralysis, *e. g.*, paralysis of the speech centre if the blow is over the left side in right-handed people, or slight paresis of the limbs of the side opposite to the injury. Most of these mild cases clear up ultimately, but occasionally the hemorrhage in one of these cases, caught in the meshes of the pia, becomes converted into a traumatic pial cyst and may lead to slight or severe epileptic focal symptoms, which ultimately may lead to severe mental deterioration. These cases are most difficult to decide as to treatment. Each case must be decided by itself; no definite rule can be laid down. Deductions must be drawn from long experience. In doubtful cases the skull should be opened and the superficial clot should be removed to avoid possible late epilepsy, and the operation of choice is a skin-bone-flap in preference to a subtemporal operation.

(b) Severe cases generally are due to rupture of the middle meningeal artery or one of its branches, or from a vessel in the dura. The hemorrhage may be beneath the dura (between dura and brain) or outside the dura (between skull and dura). Occasionally both varieties are present in the same individual, although this is not common, and these severe cases usually are associated with fracture usually of the linear sort. The symptoms vary a great deal. In the classic cases, oedema of the brain occurs, followed later by hemorrhage. In such cases, there is, after the injury, immediate confusion, irritation, or unconsciousness, followed by a nearly normal interval, again followed after a variable time, from a few minutes to sometimes twenty-four hours, by increasing stupidity becoming coma, accompanied by focal convulsions or paralyses. In other cases, the immediate oedema leads to unconsciousness, and a severe hemorrhage occurs before the patient becomes conscious, and early convulsions or paralyses appear. The paralyses in these cases generally begin in the face, since the hemorrhage in most cases is most marked low in the skull, and as the hemorrhage rises the paralysis descends to the arm and leg. The blood pressure in these cases always is high and *increasing*.

All of these cases are operative cases. They

are fatal without operation. Many recover promptly with operation. The operation of choice is always a skin-bone-flap operation. Remove the clot, check the hemorrhage, although the vessel often is occluded at the time of operation. In these cases the bone-flap can be replaced and no permanent skull defect will remain.

(c) Severe cases without focal symptoms. These cases already have been mentioned under (b). These cases are the most difficult to diagnose of the lot. They may simulate severe concussions or laceration early, or basilar hemorrhage late. The symptoms of most value are the high blood pressure *constantly rising*, and free blood in the spinal fluid, with no escape of blood or serum from nose, mouth or ears, and no exophthalmos. These cases require most experienced and careful neurological examination. The diagnosis between these cases and basilar hemorrhage cannot always be made. Operation upon these cases is fairly successful, upon basilar hemorrhage almost always disastrous. These are the cases which offer the only excuse for unlimited operation upon unconscious head cases, which in my opinion is not proper. If operation should be done, it always should be by the bone-flap operation.

4. Diffuse basilar extra-cerebral hemorrhage, due to rupture of the sinus (fractured base). These cases often are obvious. The unconsciousness is profound and the patients cannot be roused by supra-orbital pressure. In most cases, there is an escape of blood or cerebro-spinal fluid from mouth, nose, or ears; occasionally brain matter escapes. In severe cases, exophthalmos occurs from retro-ocular hemorrhage. The pupils usually are unequal and dilated, often do not react. The blood pressure is very high (170 upwards). Choking of the disc may appear in two hours, but may be wanting. There always will be blood in the spinal fluid. In a large per cent. of the cases there is paralysis of cranial nerves (1 in 7 cases). These patients die of cerebral pressure, but as a rule the pressure (hemorrhage) cannot be checked. Hence, results of operation in these cases are disastrous. One symptom is an absolute contraindication to operation, that is non-reacting, dilated pupils. In some of these cases meningitis occurs subsequent to the fracture, especially with cases where the fracture takes place through the petrous bone into the ear, and is due to ascending infection. Hence, in such cases, at once irrigate the ear with 1-1000 corrosive, pack with sterile gauze and apply a sterile dressing which is to be changed once daily with extreme aseptic care. Most of these cases are not operative. Decision as to operation should be based on the general condition, on apparent cessation of hemorrhage, with very high blood pressure, choked disc, and reacting pupils. In some of these cases the paralysis of cerebral nerves recovers, since the paralysis is due to pressure; in other cases, it remains permanent. The facial and abducens are most commonly affected.

5. Intra-cerebral hemorrhage — subsequent to trauma — generally is associated with very severe injury both of skull and brain, and the decision as regards operation is based upon the accompanying conditions. The symptoms of intra-cerebral hemorrhage cannot be cured by relief of extra-cerebral pressure.

6. Fracture of the skull, if uncomplicated by injuries of the brain, is of little consequence. A simple linear fracture requires practically no treatment beyond rest. Compound linear fractures, when possible, must be rendered absolutely aseptic to prevent possible ascending meningeal affection. *All* depressed fractures, involving the entire thickness of the bone, simple or compound, should be elevated to prevent possible subsequent irritation of the brain and Jacksonian epilepsy.

CONCLUSIONS.

1. Uncomplicated oedema of the brain almost never requires operation.

2. Slight laceration of the brain with persisting oedema sometimes requires operation to prevent cerebral degeneration. Subtemporal method is the best operation.

3. Slight extra-cerebral hemorrhage seldom requires operation; occasionally demands operation to avoid cyst formation; the operation of choice is always skin-bone-flap.

4. Extensive extra-cerebral hemorrhage, from rupture of the middle meningeal artery, with or without focal symptoms, *always* should be operated on, and always by skin-bone-flap method.

5. Basilar hemorrhage seldom can be cured by operation. When operation is done it should be done on selected cases, for relief of pressure, and usually by skin-bone-flap method.

6. Intra-cerebral hemorrhage cannot be cured by operation, although pressure may be relieved.

7. Fracture of the skull, by itself, is of little importance except when compounded, when it may produce meningitis unless made aseptic. All depressed fractures involving the entire thickness of the skull should be elevated.

DISCUSSION.

DR. W. E. PAUL, Boston: It may be stated that if the diagnosis of skull fracture is established with severe symptoms, operation is warranted and in most cases advisable. It seems desirable to be conservative and permit individuals with conditions favoring recovery to escape the bone deficiency and scar.

What are the conditions that indicate no operation, or a delay in operating? Chiefly they are the lack of severe degree of disturbance of consciousness with a good and prompt recovery of clear mentality; a lack of headache or a speedy subsidence of headache; no paralysis; a lack of injury to

various cranial nerves; absence of sepsis; normal temperature; the continuance of normal blood pressure and fundi oculi; a clear spinal fluid; a satisfactory general condition of the patient.

These considerations amount to a restatement in different form of the trite, but always dominant dictum, that the bone fracture is of little importance as a bone injury; the brain injury is the vital factor in the matter of diagnosis, prognosis and treatment.

It is perhaps not going too far to say that many cases have been called fracture in which no fracture existed. The diagnosis is blind at times, with only suggestive rather than out-and-out symptoms. Without signs and symptoms pointing to injury of the brain, and in the absence of unfavorable progress, it seems wise to advocate watchful waiting rather than be too radical about operating.

It is perhaps well to consider the nature of the injury in measuring the need of operation. The late Dr. Mumford called attention to the fact that in a series of cases at the Massachusetts General Hospital, studied by him in 1893, fractures from falls were fatal in 50% of instances; and railway fractures had a mortality of 74%; while fractures from blows on the head usually recovered. Dr. Walton has been accustomed to say that head injury from a locomotive accident he considered serious even if the symptoms were not alarming in the immediate time after the accident.

The treatment of skull fractures by daily lumbar puncture is sound theoretically and its practice has seemed favorable in the hands of some who have resorted to this measure. It would be a desirable measure to practise in the less urgent cases and would not interfere with a trephining operation later.

In fractures of the skull from direct blows operative procedure is desirable usually at the site of the blow. Fragments of bone are to be removed and the subdural space drained and freed from blood. These cases do well or ill according to the amount of parenchymatous injury. With much laceration of the brain and multiple and deep hemorrhages, a fatal outcome is likely, even though the intracranial pressure is relieved.

Such cases also are more likely to be followed by epileptic phenomena, a complication which is probably lessened by operation.

THE TEMPERATURE AS A VALUABLE GUIDE TO DIAGNOSIS, PROGNOSIS AND SURGICAL TREATMENT IN CRANIO-CEREBRAL TRAUMATISMS.

BY J. W. COURTNEY, M.D., BOSTON.

ALTHOUGH many of the surgical textbooks still in use waste much valuable space in discussing the various sites and characters of the skull fractures which may accompany traumatic injuries of the brain and its membranes, the modern surgeon and neurologist who come together in council over a given case of head injury, spend but little time on this aspect of the problem before them. The menace of a skull fracture is twofold: By its site it may invite infection, and by its character it may create serious brain irritation or compression. But, after all, the indications for dealing surgically with these contingencies have long since been crystal clear.

The real problem which challenges the combined acumen of surgeon and neurologist is to determine the nature and extent of the havoc immediately wrought upon the cranial contents by the devastating onsweep of a traumatizing force which has but partly spent itself upon the bony container.

In a very small percentage of cases this force may cause merely a concomitant tear of a meningeal vessel or of a sinus to which the fractured bone formerly afforded a sheltering groove. But in the great majority of cases its pathologic effects are in a crescendo scale whose most intense note is represented by an actual tearing of brain substance. This laceration is most commonly observable, as you very well know, in the basal surface of the frontal and the tips of the temporal lobes. And its frequent appearance in these sites is easily intelligible when we consider the scabrous character of that portion of the skull base upon which these brain parts rest.

As a less intense, but, in many cases, no less sinister manifestation of the action of the traumatizing force in question, we encounter in brain or membranes, or both, the condition which we designate *contusion*. Here the clinico-pathologic picture is clear-cut and unforgettable. Everywhere within the contused zone the cortical vessels are injected, the surrounding tissues edematous. Caught within the meshes of the pia there may be a thin glaze of hemorrhage or a more bulky bulla of transudate. Deep within the brain substance itself innumerable tiny terminal vessels are filled with thrombi, and everywhere is the inevitable secondary edema which may be sufficiently extensive to make the entire brain seem like a great sponge filled with fluid.

Such are the important intracranial pathologic possibilities which surgeon and neurologist must face in practically every recent case of cranio-cerebral traumatism. Obviously, then, in a given case there must be the clearest possible determination of the nature, extent and site of the lesions present before any operation other than elevation of depressed bone or the removal of osseous fragments is considered.

For such understanding even the most painstaking and complete neurologic examination is, for many reasons, far from adequate. In intracranial traumatisms there is a fallaciousness about the apparently obvious in neurologic symptomatology that is encountered, as far as I know, in absolutely no other pathologic condition of the cranial contents. One not uncommonly observes, for example, particularly in young subjects, a monoplegia or hemiplegia, due to circumscribed pial edema, whose mode of onset is indistinguishable in point of time and, for that matter, in every other way from that caused by rupture of the middle meningeal artery. The paralysis is identical in the two

conditions, likewise the behaviour of the pupillary and other reflexes. And yet it is vitally important, surgically, to be able to determine definitely whether the underlying cause of the paralysis is blood or transudate.

The error most commonly committed by neurologist and surgeon alike is to allow their attention to become engrossed with phenomena which spring from disturbance in or destruction of functionally well known areas in cortex or elsewhere, while they overlook the supremely important fact that lesions identical in kind with those responsible for these phenomena may be wrought at the same time by the same force in widely scattered *silent* regions of the encephalon. This is a point upon which neither too great nor too frequent emphasis can be laid.

Personal experience leads me to declare dogmatically that, of all the diagnostic and prognostic manifestations of intracranial traumatisms, the temperature throughout stands supreme in the scale of importance. Pulse and respiration are, to be sure, not negligible quantities, but I think I may say that a depression of cardiac force is no more significant here than in other forms of injury or disease. Speaking generally, with regard to the pulse in the injuries in question, it is entirely justifiable to say that a practically normal or a full and relatively slow pulse is encountered as often in recovering cases as in those which turn out badly. As to respiration, it is usually a most unreliable measure of the degree of danger that menaces, except in initial shock or in the closing phase of a fatal case. On the other hand, for precious indications, both of a diagnostic and prognostic order, one may confidently look to the temperature.

Let us study first its behaviour in intracranial hemorrhage. The immediate effect of this, as of other forms of traumatic intracranial lesions, is, as every observer knows, to produce shock, with its concomitant subnormal temperature. Unless, however, the bleeding is very extensive and complicated by co-existing alcoholic intoxication, the temperature soon regains the normal and is seldom elevated more than a single degree throughout the entire course of the case, provided no extensive degree of contusion and no degree whatsoever of laceration be present.

In uncomplicated contusion the range of the temperature is equally distinctive. In the generality of cases it rises quickly from the subnormal point caused by shock and may go as high as 102. Almost from the beginning its recessions are striking. On the morning of the day following the receipt of the trauma it may drop to 99, or even to normal, only to rise again in the evening to 101 or 101.5. The next morning may find it at 97.5; the evening at 99, or even 100. Throughout the whole duration of the case its behaviour is consistent. In favorable cases the evening range gradually narrows to the normal point, the morning recession to a point but slightly subnormal. Toward the end, in unfavorable cases, temperature and pulse rise together and may reach a very high point.

In brain laceration the initial fall of temperature from shock is of extremely brief duration. It makes little practical difference whether the tearing of brain tissue occurs in alleged heat-producing centres or elsewhere, or whether this tearing is associated with a considerable hemorrhage. In every case of laceration, regardless of surrounding conditions, the almost immediate trend of the temperature is steadily upward. And in severe cases it may rise to 104 or 105 in a few hours. In general it does not rise so quickly to this height. There may even be, in the course of a few days, recessions, but these are infrequent and insignificant. The fact remains that an early and persistently high temperature after cranio-cerebral traumatism invariably denotes brain laceration.

In the later complications of intracranial injuries—notably in arachnitis and abscess—the behaviour of the temperature is equally constant. As a preliminary to what I have to say on this phase of the problem before us, I desire to state categorically that the occurrence of chills during the course of a case of head injury is decidedly not an indication of pus formation. Curiously enough, chills are not at all uncommonly observed during the course of a case of practically uncomplicated contusion, while in arachnitis they are mainly conspicuous by their absence.

The development of an arachnitic process is, in general, seldom discernible for several days after the receipt of the trauma. Its onset is marked by a sudden jump in the temperature accompanied by such signs of cortical irritation as delirium, somnolence or great motor restlessness. The initial rise may be as much as 6° in a few hours. This more or less sudden and sharp elevation occurring at the epoch above indicated is, in and of itself, distinctive. Even more so, however, are the curious fluctuations in temperature which succeed it. Not only from day to day, but from hour to hour are these observable; and there may be a difference of 4° in the highest and lowest points noted in the space of twenty-four hours. The average temperature may be as high as 103° plus.

In cerebral abscess the behaviour of the temperature may be broadly outlined as follows: It is normal during a sinister period of quiescence which follows the antecedent activity of the process, whatever it may be, that prepares the soil for the growth of pyogenic organisms, and *subnormal* after an abscess has attained sufficient proportions to cause local manifestations of its presence. One must not, however, adhere rigidly to this formula in all cases. In certain cases development of an abscess may take place before the signs of an antecedent process have disappeared. In this event the temperature may continue high to the end.

Here let me make it very clear that I do not

claim, in the foregoing, to have covered, in every detail, all the pathologic changes which may result from trauma, or that the temperature in those described unswervingly follows, in all cases, the course set forth. What I do contend, however, is that the changes described are those of most practical importance and that in them the behaviour of the temperature as outlined is sufficiently constant to constitute a guide of such marked value in diagnosis, prognosis and treatment that, by comparison, the signally illusory indications furnished by neurologic examination alone are absolutely negligible.

Equally signal is the service rendered by the temperature in establishing a prognosis and in the adoption of rational treatment. When the chart indicates that a given brain compression is due to a *hemorrhage* which is practically uncomplicated, we know that prompt ligation of a bleeding vessel and the turning out of a clot will doubtless save a patient's life. When it indicates *contusion*, we know that we are dealing with the most insidious and treacherous pathologic process in the whole domain of cranio-cerebral traumatisms—the one, *par excellence*, in which the prognosis must always be guarded, even in cases in which the purely neurologic manifestations are of least sinister omen.

Everyone who has followed attentively the course of events in cases of cerebral contusion knows that only in few—and these of the mildest—can the danger to life be safely considered over before the end of the second week. With disturbing frequency we see cases of this order run along for ten or twelve days with but little or no psychic disturbance and with absolutely no paralysis, then suddenly become hemiplegic and pass out in profound coma. In young subjects hemi- or monoplegic manifestations due to circumscribed pial transudate disappear spontaneously. Trephination for relief of edematous compression in adult subjects is, for the most part, a fruitless expedient.

In connection with *laceration* the trephine is not even to be thought of. This condition is not invariably fatal, however, and even a temperature of 105 does not preclude recovery. In favorable cases convalescence may be painfully protracted and the temperature may for a long time stand at 99 plus. The outlook in *arachnitis* is dark in every case. In *abscess* it hinges largely upon the matter of accessibility. Apropos of operation in connection with abscess, I am again reminded of the subject of the temperature, and take this occasion to state that directly an abscess is evacuated, the temperature is apt to rise a point or two and remain somewhat elevated for 24 or 48 hours.

As I have proceeded with my exposition, it must have become increasingly obvious to those who have favored me with their attention that, while close observation of temperature behaviour in cranio-cerebral injuries markedly broadens our understanding of the intimate nature of their underlying pathology, it at the same time automatically betrays the extreme narrowness of the field of indications for legitimate surgical intervention in these injuries.

In a paper on traumatic cerebral edema* written nearly twenty years ago, I expressed my views on the surgery of cerebral traumatisms as follows:—

"Our knowledge of cerebral localization and function has been derived in part from experiment and in part from induction based upon the pathologic findings in cases of brain neoplasms, whose clinical expression has been accurately observed. In the majority of such cases the march of symptoms is slow and the portions of the brain, outside of those immediately concerned in the growth, healthy. In animal experimentation, moreover, where certain portions of the cortex have been successfully removed, this has been done piecemeal, and the remaining *healthy* portions of the brain not traumatized. In these two facts lies the secret of the limitations of cerebral surgery. In the removal of neoplasms the most brilliant successes have always attended those cases where only limited regions of the cortex and its underlying tissue were involved. In the surgery of cerebral traumatisms the most successful results have been obtained when the conditions underlying the immediate injury to the skull have most closely simulated those of a circumscribed cortical neoplasm, namely—uncomplicated middle meningeal hemorrhage."

In the nineteen years and more which have elapsed since the above was written, my views on this subject have undergone no material change. And I am today firmly convinced that every other experienced neurologist, if he be honest with himself, will frankly admit that, for the most part, the trephine is a pitiably crude and unavailing weapon with which to combat the menace of death that lies in the types of injury considered in this present paper.

INDICATIONS FOR LAMINECTOMY IN FRACTURE OF THE SPINE, WITH CORD SYMPTOMS.

BY JOHN B. HARTWELL, M.D., BOSTON.

THE status of laminectomy in spinal fractures with accompanying cord lesions is still undecided. It has enthusiastic proponents who believe that operation is indicated in every instance, and vigorous opponents who believe that operation is never indicated. The argument influencing the former is well stated by Walton[1]: "The benefit of doubt should be given to an operation which has proved at the worst neither materially dangerous to life nor detrimental to comfort, and which at least may be expected to lessen pain, to reveal the nature of the lesion with which we are dealing, and to place the cord in the best position for restoration of its func-

* Traumatic Cerebral Edema; Its Pathology and Surgical Treatment; A Critical Study. Courtney, J. W., BOSTON MEDICAL AND SURGICAL JOURNAL, 1899, Vol. cxl, pp. 345, 373, 397.

tion, where such restoration is possible; and if we may add to these comparatively modest claims the hope that an occasional life may be saved, and an occasional patient rescued from helpless invalidism of the most distressing character, should not the burden of proof be laid upon conservatism?" The conservative's reply is well made by Thorburn:[2] "In man we can't hope for anatomical recovery; vicarious conduction may allow of restoration of function to some extent, but in the case of complete transverse lesion in which vicarious conduction is manifestly impossible, no recovery whatever will take place. I have not satisfied myself that there have been any successes as regards recovery of function, save such as may be attributed to the regeneration of nerve roots only, or to the natural recovery of a cord which was but slightly injured. And further, if it were shown that in one or two instances among the two hundred published cases there had been a definite improvement or recovery, I should be inclined to regard such as the sequel of some error in the original diagnosis, rather than to invalidate a rule based upon such extensive premises."

Before we take our position in the controversy let us briefly consider the structures involved in injury, the mechanism of injury, and the results of such injury, in order that we may remove certain popular misapprehensions.

The vertebral column may be likened to a sinuously curved segmented tube, and though the sum total of motion between the segments is considerable, the motion permitted between any two segments is very small. The freest motion is permitted between the third and sixth cervical segments and in the region of the last two dorsal segments. These two situations being points at which fixed and movable segments of the spine join each other, forces applied to the spine are here concentrated, and hence the majority of fractures occur in these two regions.

The spinal cord hangs suspended in this tube. Continuous with the medulla above, it usually terminates between the first and second lumbar segments of the spine, and is loosely anchored along its course by the spinal nerve roots and "during strong flexion an appreciable ascent of the lower end takes place."[3]

The cord does not fill the bone-surrounded cylinder in which it lies. The brain, on the other hand, completely fills the cranial cavity. It is, therefore, plain that the arguments for cranial decompression, which are often applied to the spinal cord, should not apply because of the different anatomical relations which they bear to their respective bony envelopes.

Though direct violence may crush vertebral laminae and drive bone fragments into the cord, or by narrowing the spinal canal cause a compression of the cord, this is not the usual mode of injury. The common mechanism consists in a sudden, acute hyperflexion of the spine, secondary to a force applied at one or the other extremity of the column. By this jack-knifing, the cancellous vertebral bodies are made to bear the bulk of the strain, and they crush. If the force continues, the inelastic bony posterior arch cracks, and lastly the more elastic intervertebral ligaments give way. As a result of this sudden hyperflexion, the spinal cord may be stretched in the less severe cases, or contused or crushed between the posterior upper margin of the body of one vertebra and the lower margin of the lamina of the vertebra above—in the severest cases. But in either case the trauma to the cord is synchronous with the fracture of the vertebrae. The damage done, the recoil follows, and bone pressure on the cord is released unless the crush of the vertebral bodies, with secondary displacement, has been so great as to narrow the spinal canal to a diameter smaller than its contained cord. Too great emphasis cannot be placed on this fact: that the damage done the cord is as primary as the damage done the bone. The medullary injury is immediate in the vast majority of cases, not of gradual onset as some writers lead us to infer.

A second fact on which I believe far too little stress has been laid is that the force that causes the vertebral fracture is so great that in at least one-third of the cases other severe injuries than the spinal fracture are inflicted on the sufferer. Among these may be mentioned skull fracture, fracture of the sternum and ribs, with and without lung puncture and hemothorax or rupture of liver and kidneys, and fractures—simple and compound—of the pelvis and bones of the lower extremities. The particular bearing which these accompanying injuries have is in connection with surgical shock, of which I shall speak later.

Though the vertebral bodies be crushed and displaced, the vertebral arches broken, and the intervertebral ligaments torn, the dura covering the cord is rarely injured. And though bleeding from the bone and venous plexus takes place, it is rarely great enough to cause compression of the cord. Likewise the intradural hemorrhage is rarely of sufficient extent so far to fill the subdural space as to cause medullary compression.

So far as the cord itself goes, Allen[4] has shown experimentally, in dogs, that following a crushing blow to the cord, there is an immediate hyperdistention of the pia-arachnoid with blood and cerebrospinal fluid; the cord itself appears pinker than normal, and on longitudinal incision into the cord substance there is "a goodly outpouring of blood and serum." Histopathologically,[5] within fifteen minutes there are hemorrhagic foci in the gray matter of the cord and a beginning edematous condition noticeable in the white matter. Within an hour the intramedullary hemorrhage has assumed much greater proportions, and the deterioration in the staining properties of the anterior cornual cells, which after three hours is very

marked, has already begun. In six hours there is very evident disruption of the cord substance. But in addition to the local damage, as pictured by Allen, Taylor[6] and Thomas[7] have shown not only that the intramedullary hemorrhage above described may extend through the cord for a considerable distance above and below the site of injury, but that similar lesions may be produced in the cord at points far removed from the site of compression.

The immediate result of injury to the cord, whether a traumatic hematomyelia or concussion, a contusion, or a complete crush, is a loss in power of conduction, complete or incomplete, below the point of injury. If the motor or sensory paralysis is not complete, if there is not complete retention of urine and feces, if the deep and superficial reflexes—excepting the plantar reflex—are not lost below the point of injury, it is certain that a complete transverse lesion of the cord has not taken place. But, unfortunately, the reverse is not true. All the signs of a complete transverse myelitis may be present and persist for days or weeks, and then sensation or motion or reflexes return, and thereby prove that first appearances were deceiving. Unfortunately, it is only by "watchful waiting" that a positive diagnosis of the extent of the cord injury can be made.

In his experimental work on dogs, Allen found that an impact of 340 gram centimeters, or less, would cause a paralysis from which the animal would spontaneously recover, but with an impact of greater force a permanent paralysis ensued, unless an early incision into the cord was made for drainage. In the latter case, an impact of 450 gram-centimeters on the cord was survived.

The same principle undoubtedly applies to man. If the squeezing is not too great, spontaneous recovery of function will take place. If the compression exceeds a certain force, no recovery of function can be expected, unless, in rare instances, incision for drainage at the primary site of injury be practised within six hours. Of course in man the degree of cord compression in any accident cannot be estimated, and we do not know that the cord of man will be destroyed to the same extent after six hours as the cord of the dog. But it is probably safe to venture that the human spinal cord is more highly developed and more sensitive to injury than the cord of the dog. And those patients who are seen with signs of a complete medullary destruction should, I believe, be considered to have sustained an injury beyond the reach of surgical help.

Shock is mentioned in practically all the literature on spinal fractures, and leads to the inference that shock is to be expected in patients who have suffered this injury. However, in a recent review of the 133 fractures of the spine treated at the Massachusetts General Hospital between January 1, 1900, and December 31, 1914, shock is noted in less than one-quarter of the cases. And those patients in shock had received other severe injuries than the spinal fracture. Uncomplicated fractures of the spine, regardless of the extent of the accompanying cord damage, do not cause shock. The natural corollary to this statement is that shock indicates other injuries—which may be obscure—and though the patients may rally from the shock, the causative injuries may rightly modify the indications for treatment. For example, in the series studied, though treated expectantly, 100% of the patients with fracture of the cervical spine with other injuries died; 80% with fractures in the dorsal region, and 70% with fractures in the lumbar region and accompanying injuries, died. Compared with these figures, there was a mortality of 70% in the uncomplicated fractures in the cervical region, and no mortality in either the dorsal or lumbar segment fractures. The large series of cases described by Gurlt,[8] Black,[9] Burrell[10] and DeQuervain[11] make no note of the injuries sustained by the patients in shock, so the above assertion finds no confirmation from them.

Laminectomy, no one will dispute, is a major operation, and carries with it a definite mortal risk. Nor is the risk so small as many would lead us to assume. Lloyd[12] stated that in 59 out of 227 cases collected by him, death may have been hastened by operation; 55 of the 218 cases collected by DeQuervain died within a few days after the operation, though he does not specifically state that the operation hastened death; in the series studied by me, 4 deaths in 35 laminectomies were attributable to the operation—2 deaths on the table, one death an hour following operation and one death from a septic meningitis.

Laminectomy does not give a better idea as to the extent of cord destruction than can be gained from the clinical signs alone. In the series of cases I studied, the condition of the cord was noted in 30 of 38 cases subjected to laminectomy, all of whom presented signs of extensive cord damage. But in 13, or over one-third of the cases, operation revealed no demonstrable injury to the cord. In 12 cases—another third—the cord was found "crushed" and the operation was, therefore, useless, and in the remaining cases the cord was described as "lacerated," "thinned" or "slightly discolored."

Fractures of the cervical segment of the spine with accompanying cord symptoms are particularly fatal, and death is almost invariably hastened by complications referable to the respiratory tract. The paralysis of the intercostal and abdominal muscles makes coughing impossible, and the mucus that collects in the trachea and bronchi can, therefore, not be expelled. Cyanosis and dyspnea appear early in these cases. They do not take ether well, and I believe that the administration of any anesthetic is absolutely contraindicated. Add to this the

possibility of traumatizing the cord and the consequent death from respiratory paralysis, and the sum of the dangers to which the patient is subjected by operation outweighs the probable benefit.

Fractures in the dorsal region are far less fatal. There is no respiratory difficulty, and the dangers of administering an anesthetic are no greater than normal, and operative trauma to the cord a few segments above the site of greatest injury will not be fatal. In the lumbar region the cord itself may escape injury, and the damage that results from compression may involve only part of the cauda equina, which is amenable to suture.

Apart from the usual cases of broken back with immediate onset of cord symptoms, there are a few exceptional cases of fracture of the spine in which the medullary symptoms either are very gradual in onset or make their first appearance some hours or days after the original injury.

There is a third class, in which the vertebral fracture was originally unaccompanied by cord symptoms, or in which the cord symptoms were very slight and of short duration (for example, retention of urine for four or five days), but in which, after a lapse of several weeks or months, medullary symptoms begin to appear and slowly increase. In these cases there is true compression of the cord, be it by bone callus or by an infolded ligamentum subflavum or by a localized serous meningitis.

Certainly an operation cannot be indicated unless there is a reasonable prospect of bettering existing conditions or of prolonging life under existing conditions. In the usual type of fracture of the spine, accompanied by cord symptoms of immediate onset, these indications should lead us in paths of conservatism rather than of radicalism, and our indications for operation should be special and not general, and we must not be unmindful of the fact that where spontaneous improvement does take place it usually does not manifest itself short of from four to ten days.

Laminectomy, I believe, is absolutely contraindicated in all cases of fracture of the cervical segment of the spine in which there is a paralysis of the intercostal and abdominal muscles, and is contraindicated in all fractures of the spine, wheresoever located, with cord symptoms of immediate onset, howsoever slight, if other severe injuries are demonstrable, or if the patient presents or has presented the signs of surgical shock.

Laminectomy is contraindicated in cases of fracture of the spine with synchronous cord injury, in which the signs point to a complete crush of the cord, until at least four days have passed—the minimum time in which spontaneous improvement can be expected to manifest itself—and if no spontaneous improvement is shown, I do not believe that operation offers any real hope.

Laminectomy is justifiable in cases of fracture of the spine, accompanied by cord symptoms which do not point to a complete cord destruction, if the patient comes under observation within six hours, and if during the period of observation the cord symptoms are progressive. In such cases laminectomy with incision into the cord substance for drainage should be practised at once.

Laminectomy is urgently and immediately demanded in those cases of spinal fracture in which cord symptoms are of slow onset and progressive, and in those cases originally free from cord symptoms, in which medullary signs make a sudden appearance.

Laminectomy is indicated in those fractures of the lumbar spine in which the cord symptoms indicate injury to the cauda equina, and in these cases an attempt to suture the crushed trunks should be made.

Laminectomy is indicated in those cases of old fractures of the spine in which cord symptoms were originally absent or slight and of short duration, but in which symptoms of pressure on the cord develop late.

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DISCUSSION.

DR. ISADOR H. CORIAT, Boston: Dr. Hartwell has presented a most interesting and valuable contribution to the subject of spinal cord surgery, while his description of the mechanism whereby the cord suffers actual injury from accident, is remarkably clear. The surgery of these conditions is not within my province, but a brief statement of the problem as it presents itself to the neurologist may be of interest.

From the clinical standpoint alone, it is sometimes very difficult to make a diagnosis of fracture, particularly if deformity or tender points cannot be demonstrated. The conditions most likely to be confused are hematomyelia and certain cases of what may be termed acute oedema of the cord, as these conditions, under certain circumstances, may present the clinical picture of a complete transverse lesion. A complete neurological examination is essential in all cord injuries, in order to determine the height and extent of the lesion and also in an attempt to differentiate an actual crush from a hemorrhage into the central canal or central gray matter by means of the sensory reaction to heat and cold. The x-ray may also be of help, although of less value than a thorough clinical examination, since cases exist in which the x-ray shows a fracture with few or no cord symptoms, or, on the contrary,

severe transverse cord symptoms may be present without the radiographic evidence of fracture.

The presence of even diminished or a few reflexes below the point of compression, or of disseminated areas in which there is no disturbance of sensation, or of an irradiation of reflexes or a contralateral reflex, is evidence that a certain amount of conduction is taking place in the spinal cord. For instance, in stroking the soles of the feet, even if the plantar reflex is absent or diminished, if there takes place at the same time a contraction of the quadriceps, this points to cord conduction, an overflow of the reflex from the point of reflex discharge.

I believe that if there is found an incomplete lesion of the cord after injury, as evidenced by the reflexes or the sensory disturbances, that a laminectomy should be done at once before further hemorrhage or oedema takes place. If there are signs of a complete transverse lesion of the cord, no harm is done by waiting 24 to 48 hours, first for the oedema to subside and secondly, because cases of acute oedema of the cord may produce a clinical picture of a transverse lesion, due to a temporary suspension of function, as in experimental concussion of the brain.

An exploratory or decompressive laminectomy is not dangerous provided there is not too much surgical shock or if there is no evidence of diaphragmatic paralysis. The latter condition may lead to pulmonary oedema with cyanosis and inability to expel mucus; and I agree with Dr. Hartwell, that the administration of an anaesthetic in these cases is contraindicated. In addition, I feel that a rapid increase in the cord symptoms is a contraindication for operation, and a cervical lesion offers a far more grave prognosis than a dorsal or upper lumbar lesion.

In a paper on some cases of spinal cord surgery which I published in the Medical and Surgical Reports of the Boston City Hospital in collaboration with Dr. Crandon several years ago, I expressed the same viewpoint on decompressive or exploratory laminectomy as I do today.

In closing, I am reminded of an interesting case of fracture of the fifth cervical vertebra, which I saw over a year ago in consultation with Dr. Ehrenfried, and which was operated on by him at my suggestion. In this case there were symptoms of a complete transverse lesion comprising everything below the first dorsal segment, with complete flaccid paralysis of both legs. The symptoms were increasing at the time operation was done, on the third day. Laminectomy showed that the cord was decidedly oedematous, partially crushed, and with a probable hemorrhage into the central gray matter. The x-ray showed a fracture of the body of the 5th cervical vertebra with displacement of the upper fragment against the cord. After this decompression the patient made a fair functional recovery, which I feel would not have taken place without an operation. He is at present able to get about comfortably with crutches. This case was particularly valuable, since I was able to follow it for a long period of time and make a series of neurological examinations.

DR. SAMUEL J. MIXTER, Boston: The question of operation in fractures of the spine is one that has never been settled and never will be, I think. It depends largely upon the feeling of the individual surgeon, that is, his feeling at the time of the operation, whether he is optimistic or pessimistic. It is hard to make a correct diagnosis. We can make a better diagnosis now with the x-ray than we could before, but that does not give us news as to the condition of the cord. The symptoms may mislead. You may have all the symptoms of a complete transverse lesion, and yet the patient may recover, and then you know that he did not have the transverse lesion. I remember seeing one case in consultation of a young boy who was shot in the back directly in the median line. He had complete paralysis, and all the symptoms of complete transverse lesion. A most unfavorable prognosis was given and operation was advised against. He recovered, and it was simply a mistake in diagnosis. Just what happened I do not know. That was before the days of the x-ray.

It makes a great difference as the reader has said, where the lesion is. If the lesion is in the neck it is very serious, however slight the damage to the cord. Now the question comes, what to do with fractures, whether to operate on them or not. The answer to that question is entirely dependent upon whether the case is a fresh one or an old one. If it is a fresh one, I think it is perfectly justifiable to operate on the case. In the instance I spoke of, the boy recovered from what was apparently a complete transverse lesion, without operation. I have operated in two cases where we thought it was complete transverse lesion, and the patients have recovered. They might have recovered if I had not operated. No one can say. I am very sure that none of the cases that I have operated on for fractures of the neck have been killed by the operation *per se*, and in these days when the operation can be done by a local or regional anesthesia, as is now done by Fraser and others, the operation is very much safer.

There is mighty little chance, in operating on a man with a broken neck, of pulling him through. Once in a while one gets well. At any rate, I think it adds little to the danger. I said years ago, and I have not changed my mind very much, that a person who did spinal surgery, surgery for broken backs, broken necks, etc., should enter on the thing without great hope as to results, and certainly with no enthusiasm, because most of the cases will not be benefited.

When Allen and Fraser showed their dogs in which they achieved such wonderful results by longitudinal section of the cord, we were very much encouraged. In talking with Dr. Fraser last week, he told me that he has seen nothing in the clinical work so far to encourage him especially. It has been absolutely negative.

The cases that should be operated on are those with increasing symptoms, whether fresh or old, or perhaps those which show in the x-ray a definite change in the lamina of the canal. Even those cases may not be helped. Wherever there is a chance of a few fibres being left, operation certainly offers the best chance and puts the cord in the best state for healing. Drainage alone helps and if a person has those few fibres, or if you think they may be there, I think it is better to operate and give him what chance there is. That is mighty little. Your statistics will be bad, but as I have said elsewhere, the man who will not operate on account of hurting his statistics is not only a coward but a knave. That is simply my personal feeling about operations on the cord. I have no joy, and very little hope, and absolutely no enthusiasm.

FRACTURES OF THE BASE OF THE SKULL AT MASSACHUSETTS GENERAL HOSPITAL.

BY W. J. MIXTER, M.D., BOSTON.

FOR the purposes of this paper all the case records of fractures of the skull, base and vertex, concussion and intracranial hemorrhage brought to the Massachusetts General Hospital since 1890 have been studied. From this mass of records has been selected every case in which fracture of the base was proven or practically certain. The records were then gone over again and all cases first seen more than two days after injury, all cases of multiple injury in which the patient died within two hours of his entrance to the hospital were culled out. This left the early cases of fracture of the base, eliminating such as were moribund when brought to the hospital and on which operation could not be considered.

For the purposes of this paper the term "operation" may be taken to include only those surgical procedures having a distinctly decompressive effect.

In 1911 the cranial work at the Massachusetts General Hospital was given as a special assignment to Dr. S. J. Mixter and Dr. John Homans. Later the assignment was given to me and, for a portion of the time, Dr. J. B. Hartwell was associated with me in the work. For this reason the periods since 1890 are divided, not strictly by decades, but with regard to this date.

FRACTURES OF THE BASE.
1890–1917.

		MORTALITY
Total number of cases	301	
Total deaths	163	54.1%
Cases treated expectantly	209	
Deaths	123	58.3%
Cases treated by operation	92	
Deaths	40	43.4%
Cases treated by operation	30.6%	

CASES DIVIDED BY PERIODS.

	1890-1900		1900-1911		1911-1917	
Total number	101		126		74	
Operations	9		29		54	
Cases operated on		9%		23%		71.5%
Total deaths	68		68		27	
Mortality		68%		54%		36.5%

INCIDENCE OF PROVEN MENINGITIS.

Without operation	7
With operation	1

INCIDENCE OF PROVEN INTRACRANIAL HEMORRHAGE.

Without definite localizing signs	42
With definite localizing signs	19

		MORTALITY
Operations performed within two days of time of injury	85	
Deaths	35	41.2%
Operations performed more than two days from time of injury	7	
Deaths	5	71.0%

AVERAGE TIME IN HOSPITAL OF CASES THAT RECOVERED.

Treated expectantly	21.8 days
Treated by operation	20.6 days

SUMMARY OF PERCENTAGES.

	OPERATIONS	MORTALITY
Total	30.6%	54.0%
1890-1900	9.0%	67.0%
1900-1911	23.0%	54.0%
1911-1917	71.5%	36.5%

These statistics may seem a bit startling and it is only fair to state that changes in the community may have a little bearing on them. For example, the building of the Relief Station in Haymarket Square has cut down the number of our severe accident cases from the City of Boston, while the development of small hospitals throughout the state and industrial accident insurance have tended to diminish the number of cases we receive from the railroads. On the other hand, the increasing use of automobiles tends to bring the number up. I have the impression, however, that we are not getting quite as large a number of desperate cases as we did when I was a house officer. It is also probable that we are now more able to prove a diagnosis of fracture of the base than we have been in the past.

These tables show a fairly constant number of cases throughout the past 27 years but a very marked decrease in mortality associated with a marked increase on the number of decompressions performed.

A fact of great interest, and one on which I wish to lay especial emphasis, is the relatively high mortality in late operations. I wish also to call attention to the fact that massive hemorrhages were found at autopsy or at operation in 61 cases, and that localizing neurological symptoms were lacking or confused in 60% of this number.

The comparative rarity of proven meningitis was a great surprise to me as I had expected to find quite a large number of cases showing definite evidence of meningeal infection. Doubtless there were a good many other cases which had meningitis but it was not shown in the records.

I shall not go into the indications for operation in fracture of the base as that is covered by Dr. Nichols' paper, but shall state briefly our usual routine on such cases when they come to the accident ward.

At entrance a careful examination is made, including urine and blood pressure, and the history obtained from bystanders, police, etc. A half hour pulse record is started, x-ray taken of the head and, in most cases, lumbar puncture is done.

As a rule, the decision for or against operation can be made at once, but in a certain number of doubtful cases it is necessary to wait from 6 to 24 hours in order to observe the increase or decrease in symptoms. If the decision is made

to postpone operation or not to operate at all, the patient is sent to the ward and the pulse, temperature and blood pressure watched and the condition of the eye grounds noted at intervals. Epsom salts, by mouth or stomach tube, and an enema are usually given within a few hours. The general pressure symptoms and the general condition of the patient are of fully as great importance as an indication for operation as any localizing signs.

If operation is indicated and there are no localizing signs, a right subtemporal decompression is performed as advised by Dr. Cushing. This operation gives access to the portion of the base most frequently injured, the middle meningeal artery, and permits of a fairly thorough exploration of that portion of the skull and brain. It uncovers a silent area so that if the brain presses strongly against the sides of the opening there is no resulting paralysis.

A left-sided subtemporal decompression is more dangerous as it uncovers the speech centre. If there are definite localizing signs as shown by the neurological examination, depressed bone or x-ray, the place of operation is chosen so that the portion of the brain indicated may be explored.

A moderate-sized decompression is usually sufficient, although, at times, extreme bulging of the brain makes it necessary to attack both sides. The inside of the skull is explored as far as possible, and loose or depressed fragments and extra- or intradural clots removed and hemorrhage controlled as far as possible. A rubber drain is then placed well down to the base inside the dura, which is left widely open, and the temporal muscle and scalp are closed about the drain. It is not necessary to make this closure in as painstaking a manner as in tumor cases as the bulging, as a rule, only persists for a very short time. The drain is removed at the end of 24 to 48 hours and should never be replaced.

Perhaps the most striking thing to me in the series of decompressions for fracture of the base which I have performed is the great excess of cerebro-spinal fluid under pressure, which is so frequently encountered, and the profuse drainage which occurs for a day or two. This increase in cerebro-spinal fluid is found only in the early cases, whereas in the old cases (48 hours or more after injury) the brain itself seems edematous, bulges strongly into the opening and effectually prevents any subsequent drainage of cerebro-spinal fluid. I take this to be an added reason for early operation and this is borne out by the higher mortality in the cases I have seen in private practice, a large per cent. of which have come under my care from three days to a week after injury. The late operation is much more difficult and dangerous on account of the danger of rupture of the bulging brain.

In concluding I would sum up as follows:

Decompression in the proper cases of fracture of the base of the skull markedly lowers the mortality.

Localizing signs are of value but are certainly no more important than the evidences of increased intracranial pressure.

Operation should be performed as early as possible, as late operations are much less effective.

A BRIEF REPORT OF CASES OF FRACTURE OF THE SKULL AND SPINE, FROM THE FOURTH SURGICAL SERVICE, BOSTON CITY HOSPITAL.

BY FREDERIC J. COTTON, M.D., BOSTON,
AND
WILLIAM F. COTTING, M.D., BOSTON.

THE brief summary herewith given presents simply the summary of the cases entering the fourth surgical service at the Boston City Hospital since March 1, 1916, in which there was skull fracture actually demonstrated or suspected or fracture of the spine. Fifty-one cases were listed.

The cases were taken as they came, and the series expected to make out a case in favor of certain theories held by the surgeons on this service.

This they do though perhaps in the rather cold and grudging way which is the habit of statistics. Of this more in the concluding paragraph.

First as to the skull cases:

There were thirty-eight cases so listed. Of these, four were to be subtracted—data insufficient or obviously did not really belong to this series.

There were ten that represented only the picture of mixed alcohol and concussion, only too familiar in a municipal hospital on the first two days of the week, all recovering in the usual prompt fashion, and calling for no further attention. Three were of rather severe concussion, unconscious but tending to improve, and recovered substantially in 12-72 hours. All recovered fully.

There were twenty-one cases of definite skull fracture in all; fourteen of them treated—or untreated—without operation on the skull.

1. One case showed unconsciousness and spasticity without localizing symptoms. Spinal puncture showed bloody fluid. He had fallen at the end of a prolonged debauch, was in wretched shape and died before anything was done.

2. In one other case there was obvious alcoholism, no fracture detected, no signs of brain trauma. At one time, after four days, the writer was on the point of doing a decompression for brain edema, but the man got suddenly better, only to collapse suddenly and die a couple of days later. Autopsy showed a fracture and brain laceration to reinforce the alcoholic condition. This man would have stood a better chance if decompression had been done, perhaps.

The other eleven recovered without operation, other than the cleaning out of the wound in the five that had scalp wounds: the list is appended, because these cases were *deliberately* let alone, yet several of them seem to fall within the limits of usual "operative cases."

3. Woman of 40. Had fallen downstairs; unconscious, but could be roused to mumble a few words. Pupils without light reaction. Left eye showed subconjunctival hemorrhage and marked exophthalmos. Ecchymosis about both eyes. Rather profuse bleeding from both ears and from nose. Reflexes not abnormal.

This patient had an obvious fracture of the base—anterior and middle fossae, probably a very extensive basal fracture, but bad as the condition was she seemed to be getting no worse and there were no focal signs of brain pressure. No signs of increased intracranial pressure.

Nothing was done.

In four days the exophthalmos was practically gone, she was conscious though not rational.

Twelve days after entrance she was transferred to the nerve service, irrational and noisy—apparently from alcohol, but without physical signs of any damage from the trauma.

4. Struck by falling log. Confused, not unconscious. Odor of alcohol. Unequal pupils. Orbital ecchymosis on left. X-ray showed parietal fracture. No operation. Developed acute delirium after four days, also partial paralysis of left arm and double Babinski. Blood pressure 125-100. At eleven days became entirely rational. Recovered.

5. Fell on ice. Irrational. Lacerated wound of eyebrow—stellate fracture beneath it shown in x-ray. Reflexes sluggish. No operation. Recovered.

6. Boy of 6. Fell down two flights of stairs, striking back of head. Semi-conscious. Hematoma over back of skull. X-ray showed occipito-parietal fracture. Marked stiffness of neck (x-ray shows this to be due to neck fracture). What was especially interesting was the result of the eye examination by Dr. Williams, who found beginning choked disc. Dr. Fairbanks' neurological examination found no focal signs and no signs of intracranial pressure other than the choked disc.

We decided to await developments.

The choked disc presently subsided and save for the neck damage the child was entirely well after three weeks.

7. Woman. Wound from right eye to ear. Fracture right parietal shown by x-ray. No focal signs. No operation. Recovered.

8. Fell 15 feet. Unconscious. Scalp wound left parietal. Pupils dilated. Knee jerks exaggerated. Respiration slow, stertorous. Pulse 100. Two days later delirious. Temp. to 104. Normal two days later. X-ray shows parietal fracture. Speech thick for a time but cleared. Entirely well at fifteen days.

9. Woman. Fell down stairs. Subconjunctival hemorrhage both sides. No symptoms save dulness. Well at eighteen days.

10. Boy of nineteen. Struck by car. Confused. Bleeding from nose. Profuse bleeding and loss of cerebro-spinal fluid from both ears. Pupils contracted, unequal. Abducens paralysis both eyes. (Also fracture of lower spine accounting for absent knee and ankle reflexes.) No operation on skull. Recovered showing only prolonged irritability and slow recovery of abducens paralysis.

11. Man of 52. Struck by car. Scalp wound under occiput. Comatose. Can be roused partly. No paralyses. X-ray showed fracture of the base posteriorly. Recovered without operation. Well at 12 days.

12. Fell ten feet. Wound on forehead. Fracture frontal bone. No brain symptoms. Recovered without operation.

13. Kicked by horse. Compound fracture frontal bone. Cleaned out. No other operation. Recovered entirely save for slight ptosis, persisting after eight days.

14. Boy of fourteen. Double subconjunctival hemorrhage, severe, with a little exophthalmos and some dulness mentally. Recovered completely, though the hemorrhages were a long time absorbing.

Now as to the cases operated on, the reasons, and the results.

15. Boy of eight. Sewed up as scalp wound by outside doctor. Septic on entrance. No focal signs except weakness of left hand, but an obvious fracture of the vertex. X-ray showed a plate of at least an inch depressed for ¾-1 in. Kept six days until the wound was clean. Then operated on. On removal of the fragment there was terrific hemorrhage from the torn longitudinal sinus which could not be picked up in the mess of torn brain tissue, but was readily controlled by packing held in by suturing flaps over it. Four days later this pack was removed. He did perfectly well except for slight paresis of the one hand, clearing slowly when he was discharged.

16. Boy of two. Fell three stories. Semiconscious. Left arm with spastic paralysis. Left leg distinctly paretic with knee jerk increased on left, and left Babinski. Systolic pressure 80.

We waited on this case, but while the power partly returned to the left leg and the arm was no longer spastic, yet the arm was paralyzed still and the mental condition did not clear.

Accordingly, a right-sided parietal decompression was done. Brain found under slight pressure. On opening the dura there was prompt improvement in both pulse and respiration.

Within two days he became, and thereafter remained, free of all brain symptoms.

17. Small boy. Struck on head with a shovel. Semiconscious. No localizing nerve signs. Compound fracture over left parietal region with depressed fragment. Immediate oper-

ation. Removal of fragment, drainage of torn brain tissue. Next day developed paralysis of left arm and face. On the fourth day blood clot removed from under the sutured flaps. At eight days remained only some weakness of the hand.

18. Aged 12. Fell from a truck, on his head. Admitted unconscious. All reflexes increased, left more than right. Double Babinski. Slight bleeding from the nose. Spinal tap showed bloody fluid. Fracture evidently basal. On the fourth day Dr. Thomas advised decompression. This was done on the ninth day as he did not clear up fast enough. A typical left parietal decompression was done. Brain not pulsating until dura was opened. No blood or clots found.

Four days later he was conscious and rapidly became rational though continuing to be irritable. At twenty days he went home well.

19. Fell down stairs. Alcoholic. Two wounds left occipito-parietal, right parietal with depression of bone under the latter. Irrational. No localizing signs at first, but after three hours whole left side became paralyzed. Knee jerk absent. Babinski present on the right side. Next day operation showed very large epidural hemorrhage which was cleared out and the cavity packed. The paralysis disappeared promptly and entirely. Unfortunately he died after a week of erysipelas of the face—not from the wound.

20. Aged 17. Struck by automobile. Semiconscious. Compound fracture right occipital low. No focal signs. Blood pressure 108-80. Some bleeding. Immediate operation to remove fragments and clot. Drained for four days. Sepsis developed, very slowly. At eleven days wound explored and cleaned out. Five days later on account of temperature the wound again opened up—deeply. No pus found. Lumbar puncture at twenty days showed streptococcus in the fluid. He developed a brain hernia, became comatose and died on the 24th day.

21. Aged 11. Struck with an axe on left parietal region. Brain oozing. Conscious. No focal symptoms. Immediate operation. Removal of small fragments and clot. After five days scalp wound showed sepsis. A week later developed left internal strabismus. After 23 days a second operation showed pus beneath the dura. At 50 days third operation showed considerable abscess deep in temporal lobe. Ten days later developed brain hernia and bilateral exophthalmos and died on the 65th day.

It will be noted that of the seven deaths in this series, two were from wound sepsis, one from facial erysipelas, one in a case beyond hope to start with, and only one from causes that might have been mitigated by operation.

Stated in another way, of 21 cases of proved skull fracture, only seven were operated on; of these there were no deaths following operation save by way of sepsis—late; while of those not operated on there was one death in a hopelessly smashed case and only one death from *alcohol* combined with trauma in which operation after 3 - 4 days would perhaps have been better.

It has been the service rule *not to do radical operations early,* just to clean up compound damage and to await results.

About fifteen years ago the rule was prompt decompression, and the mortality was frightful.

About four years ago this scheme was revived and still has a vogue not as yet fully checked by the results—which have been very bad so far as seen.

If one operates early there is a large percentage of deaths, occurring with curious accuracy 10 or 12 hours after operation, from a combination of shock, uncontrollable leakage of blood and of cerebrospinal fluid.

Certain cases like that one of the subdural hemorrhage just cited, growing steadily worse from hemorrhage, must be operated on and take their chance.

In the main, however, if we can wait,—and we mostly can wait safely from 48 - 72 hours,—then the bleeding has ceased, the shock is over, and we can operate or not—mostly not, without much risk and for definite reasons.

Depressed fragments, signs of cortical pressure, persistent slowness in clearing of the mental state, with or without signs of actual pressure, are the indications and the only indications.

In the early stages, particularly in base fractures, intracranial pressure is not the sole trouble; when they die it is apt to be from an operation which renews bleeding as it removes pressure, a bleeding from sinuses or little basal veins that we cannot control, which with the added trauma is enough to turn the scale.

Of the spine cases, fourteen in all, three were fractures of the transverse processes only, hardly worth discussing here, interesting only as to whether this fracture amounts to anything as to prognosis or is a negligible factor.

One was of the not unfamiliar type of compression or flexion fracture of the first lumbar vertebral body, with signs of nerve pressure but no cord involvement—a condition never in itself dangerous to life, though requiring mechanical treatment and real after-care to ensure recovery of satisfactory function.

Three cases, oddly enough, were fractures of the axis, without signs of cord lesion or of nerve pressure, but with very rigid necks in all three. Two were men, one a small boy of two years. All three were discharged in good condition with adequate mechanical support.

There were four neck fractures with cord damage.

Case 1. A boy of ten had fallen off a team on his head. He had a *loose* neck fracture, possible skull fracture, and so feeble a pulse as to pre-

clude any chance of operation. He died within three hours.

CASE 2. Aged 48. Was seen the next day after his fall. Symptoms had begun only the morning of admission,—numbness and weakness of arms. Neck tender and spastic. X-ray showed fourth cervical forward on the fifth. Three days later the paresis of arms was increasing. On the fourth day the luxation was reduced, apparently completely, under ether. Paralysis not improved. Died suddenly, absolutely without prodromal signs, next day.

CASE 3. Sensation gone from waist down. Reflexes gone. Motor paralysis of legs complete, partial of both arms. Priapism present. X-ray showed fracture luxation of the 6th cervical forward on the 7th.

Laminectomy. Arches of 6th and 7th removed.

Motion in left arm improved and he recovered some motion in legs. Distended abdomen a serious factor, some vomiting. At five days his breathing became stertorous—the pulse failed gradually and he died.

CASE 4. Fell down stairs. Fracture luxation of 4th on 5th cervical. Neurological examination by Dr. Knapp showed a crossed paralysis. Motor paralysis of right arm and leg. Analgesia and thermo-anaesthesia on the left with hyperaesthesia. Lesion unilateral, right, about the 6th segment.

Laminectomy—Cotton—at three days.

His sensation became normal and when discharged six months later he could walk without help and, despite contractures, had regained a good deal of power in the hand.

There were three cases of dorso-lumbar lesion.

CASE 5. Boy of 19. Also had a skull fracture. Not seen until two weeks after injury. Obvious lesion just below dorsal lumbar junction. Retention urine. Total loss of power in left leg. Some in right leg. Reflexes absent. Sensation diminished on perineum, about buttocks and over outer sides of feet. Seen by Dr. Knapp before entrance; operation not advised. Ten days after I saw him, I could see no great improvement, and with Dr. Knapp's approval did a laminectomy, removing arches of the 1st, 2d, and 3d lumbar. After three weeks he began to improve slowly.

Now, after nearly a year, he can walk without support but has some loss of power in the peroneal muscles—some flexion contracture of one knee which is improving. There is some loss of sensation of sacral region. Sensation in perineum and sphincter power are normal. Improvement continues.

CASE 6. Fell off roof. 60 feet. Usual picture of cord crush—probably total about the 8th dorsal.

Laminectomy—cord found completely crushed. After operation vomited and grew weaker. Died at four days.

CASE 7. Fell four stories. Paraplegia, priapism, etc. Upper dorsal laminectomy three days later done because he began to lose power in arms. Died immediately after operation.

There is probably no definite moral to these cases, except what we have long known; first, that cases without cord lesions belong in a class of their own, that apparently total cord lesions, if operated on, should be treated merely as a justifiable gamble, with death certain the other way, and that cases of cord lesion, operated or not operated, have a disconcerting way of going to pieces slowly, or dying unexpectedly in no precise concord with our ideas of the pathology of the given cases.

Two cases of laminectomy given here show really worth-while results and should constitute a real argument for doing operations on the serious cases that obviosuly do not involve complete severing of the cord, in the absence of satisfactory improvement after a few days.

DISCUSSION.

DR. E. A. CODMAN, Boston: I should like to take exception to one statement that Dr. S. J. Mixter made, and ask him if he would not modify it. I refer to Dr. Mixter's statement that anyone who considers his statistics in such operations is either a coward or a knave. My idea of the best possible surgeon in these cases would be the one who, if he did not believe the operation would be successful in his own hands, would get Dr. Mixter to do it. Dr. Mixter is in the position that he speaks of, but I do not feel that the rest of us are. A man who stands at the head of his profession here in Boston, and is president of the American Surgical Association, must operate on the very worst set of cases. Dr. Jason Mixter is in the same unfortunate position because he has had, at the Massachusetts General Hospital, one of the best opportunities to study fractures of the skull that any surgeon has ever had.

Personally, at any rate, I do not want to operate any case of skull fracture unless I am very sure that it is going to come out right. I should certainly consider my statistics unless both Dr. Mixter and his son are away on vacations.

DR. E. R. NEWTON, Somerville: I should like to ask Dr. Cotton in regard to the cases with complete recovery in such a short time, what was the future outcome? In a few cases where they have apparently fully recovered, then we hear a few months afterward that they drop dead suddenly—wherein those cases of injury of the spine and skull do not give us good results. We ought to follow them up well and get the absolute end-results. I think that is a very valuable thing. I do not know where the time limit comes, but if a person dies some months afterwards and an autopsy is made, it is only with the knowledge of these cases that we can depend upon what the results were. Still, I do not think that may influence the question of operation, which seems to be the point here.

When you hit statistics you hit Dr. Codman. He is the statistician today of the surgical end of hospital work, and we all admire his work in that line. Statistics, however, may do as much injury

as they do good. If you are handling an individual case and depending upon a lot of other cases you are sometimes led away from your individuality and your individual consideration of that particular case.

It seems to me also that Dr. Mixter hit a very nice point when he spoke of optimism and pessimism. A good deal does depend surely upon the individuality of the surgeon.

Discussion closed by DR. F. J. COTTON: These were just a year's cases. I have no statistics, but I can say that whether you have bad skull fractures or minor ones, there seems to be a lack of tolerance to alcohol in these cases, after recovery, and the late end results are very largely influenced by that. That, of course, tends to invalidate any statistics.

Clinical Department.

CHOLECYST-DUODENAL FISTULA.

BY EDWIN D. GARDNER, M.D., NEW BEDFORD, MASS.

THE following case of cholecyst-duodenal fistula seems of sufficient interest to be recorded:

F. H., female, 60 years old, single.

Family history. Negative.

Past history. Typhoid fever when a child. Has had four attacks similar to the present. First attack at age of 21. Last attack, nine years ago, was very severe, with much pain and high fever. Her physician at that time told her that she had pus in her gall-bladder and that it might rupture. She refused operation. Has had jaundice, chills, clay-colored stools and dark urine with all attacks. Past history otherwise negative.

Present illness. Seventy-two hours ago had very sudden and very severe pain under the right costal margin. Pain was constant and knife-like and radiated to the right shoulder blade, and also through to the back. Nauseated, and vomited dark green fluid repeatedly. No blood seen in vomitus. Bowels very constipated. Forty-eight hours ago became deeply jaundiced. Feces clay-colored and urine greenish in color. Twenty-four hours ago all pain stopped and jaundice began to clear up rapidly. Six hours ago pain came on again and patient is now having attack like first one, only less jaundiced.

Physical Examination. Skin and conjunctivae moderately jaundiced. Heart slightly enlarged but apparently compensated. Loud systolic murmur heard all over praecordia, loudest at apex, transmitted to axilla. Abdomen level, tympanitic throughout. No masses felt. No costo-vertebral tenderness. Kidneys and spleen not felt. Marked rigidity of the muscles on the right side of the abdomen, especially in upper quadrant. Spasm of muscles on left. Marked tenderness in the epigastrium and just below the right costal margin in region of gall-bladder. Could not feel any masses because of rigidity. No dulness in flanks. Liver dulness fifth rib to 1 cm. below costal margin.

Blood pressure 125. The urine was negative, except for the presence of bile. Stools clay-colored. White count 16,000. Temp. 101.6; pulse 100. Respirations 22. It seemed to me to be probably a cholecystitis and, because of the marked rigidity, I was afraid of empyema of the gall-bladder.

At operation I found a mass of adhesions in the region of the gall-bladder—adhesions tying up the liver, gall-bladder, stomach, duodenum and omentum. The gall-bladder was small and apparently mostly scar tissue and not particularly inflamed. Running from the fundus of the gall-bladder to the duodenum was a fistula about the size of a lead pencil, and embedded in the fistula, about one-half inch from the gall-bladder, was a stone about the size of a large pea. This I milked into the gall-bladder. The common-duct region was a mass of dense fibrous adhesions, so that it was hopeless to find the common duct. I felt that possibly the common duct was obliterated, so I did not think it wise to remove the gall-bladder and destroy the fistula. Drainage of the gall-bladder might mean a duodenal fistula, so I removed the two stones, and, as the gall-bladder apparently was not acutely inflamed, sutured it up tight, closing with great care. Immediately after the operation all went well. The temperature dropped to normal, the jaundice cleared up rapidly. On the fifth day the temperature rose to 100.4 and the patient complained of pain in the right chest. Examination showed a lobar pneumonia. An internist saw the case with me and agreed with my diagnosis, but also felt that there was fluid in the pleural cavity. X-ray showed a pneumonia on the right side but no fluid, and the diaphragm was pushed up on that side. Chest tap was dry. On the ninth day, the wound discharged profusely a thick, foul pus, which later became thin and excoriated the skin. This discharge kept up for about a month. It seemed to me that we were probably dealing with a sub-phrenic abscess and possibly a leakage through the gall-bladder and fistula. The patient finally convalesced and went home. I am not sure now but that I should have drained the gall-bladder.

At the end of a year after the operation she reports she has had two or three attacks of pain in the epigastrium, going through to the back; also pain in gall-bladder region, with nausea and vomiting. No jaundice. I saw her in one attack. Complained of severe pain. Temperature 100.6; pulse 80. Tenderness in the epigastrium and region of gall-bladder. No spasm and no masses. No jaundice. Stools and urine o. k. This attack lasted twenty-four hours. Feels perfectly well between attacks. The right diaphragm is still pushed up, the liver dulness being from the fourth rib to 1 cm. below the costal margin. The right lung is all right. There is slight bulging of the scar and the wall feels weak at this point, but there is no definite hernia as yet.

I wish to thank Dr. A. V. Pierce of New Bedford for referring the case to me.

LONDON DEATH RATES.—The death rate in London during the month of July, 1917, was 10.9, the highest rate being in the district of Southwark, which was 15.3; and the lowest rate being in the City of London district, which was 5.2. The lowest rate among the residential districts was 8.4 in Lewisham.

Harvard Medical School.

HARVARD MEDICAL ALUMNI FUND.

ELEVENTH ANNUAL REPORT FOR THE YEAR 1916

To the Contributors of the Harvard Medical Alumni Fund.

Dear Doctor:

During this last year alumni of the Harvard Medical School have contributed $3000 to the furtherance of clinical teaching in the School. By this action the alumni have actively promoted the clinical instruction of students in five departments: Medicine, Neurology, Obstetrics, Pediatrics and Surgery. The total sum thus far contributed to teaching through the Harvard Medical Alumni Fund during the ten years of its existence now amounts to $20,200, and the Permanent Fund has reached $9,088.16.

The Alumni Assistants during the year were as follows:

Dr. George P. Denny, 1912,
Alumni Assistant in Medicine.

Dr. Gilbert Horrax, 1913*
Alumni Assistant in Surgery.

Dr. F. M. Rackemann, 1912
Alumni Assistant in Medicine.

Dr. Lewis W. Hill, 1913
Alumni Assistant in Pediatrics.

Dr. George Clymer, 1911
Alumni Assistant in Neurology.

Dr. Raymond Titus, 1909
Alumni Assistant in Obstetrics.

* Johns Hopkins.

It is perfectly plain that this present school year, 1917-1918, will make great demands upon the Medical School because of increased cost of supplies. It is a year when the School most needs our help. Many of our contributors are at the front and they should not be asked for subscriptions; therefore, despite the heavy demands upon us all from many sources, it is upon us who are still at home that the burden of supporting the alumni teachers must fall. Not only must we give as before, but we must give as well for those who are at the front. Fortunately, it has been arranged that all expense of collection of the fund this year will be eliminated and every cent collected will go to an Alumni Assistant. We hope that the alumni will bear these facts in mind when they send their gifts to the Treasurer.

Please make checks payable to E. P. Joslin, Treasurer. Address: 81 Bay State Road, Boston.

FREDERICK C. SHATTUCK, *President,*
ELLIOTT P. JOSLIN, *Treasurer of Committee of Alumni Fund,*
MALCOLM STORER,
RICHARD C. CABOT.

FINANCIAL STATEMENT.
1916

	1906	1907	1908	1909	1910	1911	1912	1913	1914	1915	1916
Amount subscription	$2,912.00	$1,975.00	$2,013.05	$2,109.00	$1,855.49	$2,364.14	$2,184.50	$2,217.50	$2,125.50	$2,173.10	$2,489.87
Int. on current funds	9.32	18.53	49.10	28.18	30.58	29.53	26.34	26.03	15.29	31.38	58.26*
Total yearly income	$2,921.32	$1,993.63	$2,062.15	$2,137.18	$1,886.07	$2,393.67	$2,210.84	$2,243.53	$2,140.79	$2,204.48	$2,548.13
Balance carried forward		1,415.79	807.08	847.00	899.58	679.15	564.37	476.41	504.84	581.80	547.57
Subscriptions paid in advance	.....	.....	.....	.....	.....	107.00	116.00	24.00	10.00	98.00	186.00
Applied for salaries	.....	$3,409.42	$2,949.23	$2,984.18	$2,785.65	$3,179.82	$2,891.21	$2,743.94	$2,655.63	$2,884.37	$3,281.70
Applied for permanent fund	1,200.00	1,500.00	1,500.00	1,500.00	1,500.00	2,000.00	2,000.00	2,000.00	2,000.00	2,000.00	3,000.00
Expenses		1,000.00	500.00	560.00	500.00	500.00	300.00	150.00	.....	150.00	.....
Balance in bank	305.53	22.34	102.23	24.60	106.50	115.45	114.80	89.10	95.74	88.80	82.89
	1,415.79	887.08	847.00	899.58	679.15	564.37	476.41	504.84	581.89	645.57	198.81
TOTAL	$2,921.32	$3,909.42	$2,949.23	$2,984.18	$2,785.65	$3,179.82	$2,891.21	$2,743.94	$2,677.63	$2,884.37	$3,281.70
Gifts to permanent fund	2,430.00	2,430.00	3,430.00	3,930.00	4,490.00	4,990.00	5,490.00	5,790.00	5,940.00	5,940.00	6,090.00
Int. on permanent fund		79.54	254.67	460.12	660.16	941.16	1,239.84	1,811.35	2,207.70	2,565.39	2,998.16
Addition to permanent fund	.....	1,000.00	500.00	560.00	500.00	500.00	300.00	150.00	.....	150.00	.....
TOTAL permanent fund	$2,430.00	$3,509.54	$4,184.67	$4,950.12	$5,650.16	$6,134.16	$7,029.84	$7,751.35	$8,147.70	$8,655.39	$9,088.16
No. of contributors	295	276	260	251	226	384	361	344	357	325	332

* Includes interest for 1917, amounting to $27.48.

CERTIFICATION OF PERMANENT FUND.

Dr. E. P. Joslin,
81 Bay State Road, Boston.

Dear sir:

On April, 1917, the President and Fellows of Harvard College held $6,090 for the Permanent Fund of the Harvard Medical Alumni Association, with interest upon the same amounting to $2,998.16, making a total of $9,088.16.

Very truly yours,
(Signed) C. F. ADAMS,
Treasurer, Harvard College.

CERTIFICATION OF CURRENT FUNDS.

To the Trustees of
Harvard Medical Alumni Fund,

Gentlemen:

Owing to a misunderstanding, the books and accounts of your Treasurer for the year ended Feb. 28, 1917, did not reach me until today. I have now carefully audited them from the date of my last audit (March 22, 1916) to June 30, 1917.

I find the accounts carefully and accurately kept, all moneys received have been promptly deposited in bank, for all payments there are satisfactory vouchers on file and the same are properly entered on the Record Book.

Respectfully submitted,
(Signed) GEO. S. CHASE, C.P.A., *Auditor.*

1842

*Taylor, O. B.
*Wellington, J. L.

1853

Storer, H. R.

1854

*Choate, David
Green, S. A.
Stone, L. R.

1855

Oliver, H. K.
*Wakefield, A. J.

1856

*Lamson, J. A.
*White, J. C.

1857

*Campbell, B. F.
Sprague, F. P.

1858

*Cheever, D. W.
*Derby, H.
Towar, G. W.

1859

*Heard, John T.
*Nichols, J. T. G.
*Shaw, H. L.
Wesselhoeft, W.

1861

Blake, J. G.
Edes, R. T.
Hubbard, C. T.

1862

*Clark, A. P.
*Wood, Albert
Wright, W. A.

1863

Cutler, E. R.
Driver, S. W.
*Greene, J. S.
*Hayden, D. H.
Perry, John G.

1864

Marcy, H. O.
*Robbins, N. A.

1865

Blake, C. J.
*Inches, C. E.
*Parsons, J. W.
*Wadsworth, O. F.

1866

Green, J. O.
Nichols, A. H.
Rice, C. H.
Warren, J. C.

1867

Kennedy, Geo. G.
Richardson, W. L.

1868

Borden, Sir F. W.
*Bowditch, H. P.
*Coggin, David
Elliot, Herbert
*Fitz, R. H.
Gay, G. W.
*Jelly, G. F.
*Pratt, H. J.
Quinby, H. M.
Wells, Frank

1869

*Draper, F. W.
*Goss, Francis W.
Shattuck, G. B.

1870

*Boutwell, H. T.
Farlow, W. G.
Hayes, S. W.
Putnam, J. J.
Spalding, J. A.
Wheeler, Leonard

1871

Blodgett, A. N.
Dixon, L. S.
Gordon, J. A.
*Spooner, J. W.
*Tinkham, G. W.

1872

Channing, Walter
Cutler, E. G.
*Fenwick, J. B.
Lovejoy, C. A.
*Mason, A. L.
Morton, W. J.
Woodbury, L. A.

1873

*Ayer, J. B.
Bradford, E. H.
Lawrence, R. M.
Porter, F. E.
Shattuck, F. C.

1874

Bigelow, W. S.
Davenport, F. H.
Emerson, E. W.
*Gerry, E. P.
*Rotch, T. M.
Thomas, F. S.
Williams, C. H.

1875

Dunn, W. A.
Nichols, C. L.
Stedman, Geo.
Stedman, H. R.
Whitney, W. F.

1876

*Cabot, A. T.
Huntington, T. W.
McCormack, C. J.
Putney, G. E.

1877

Bancroft, W. B.
Booth, E. C.

1877 (Cont.)

Daniels, E. A.
Everett, O. H.
Farlow, J. W.
Green, C. M.
Howe, O. T.
*Hutchinson, Marcello
Lopez, Rafael
Peters, E. D.
*Richardson, M. H.
Sheldon, C. C.
*Swift, J. B.

1878

Bacon, J. E.
Brannon, J. W
*Bryant, John
Comey, P. P.
Elliot, J. W.
Kilby, H. S.
Leland, G. A.
Mason, W. C.
Minot, J. J.
Morse, H. L.
Tuttle, G. T.
Walker, C. R.
Woodward, S. B.

1879

Bowditch, V. Y.
Bowers, W. P.
Broughton, H. W.
Hun, Henry
Mixter, S. J.
Prince, Morton
Swartz, G. T.
Wheeler, J. B.

1880

Briggs, E. C.
Dewey, C. A.
Grandin, E. H.
Jaques, H. P.
Pierce, M. V.
Ryder, Godfrey
Walton, G. L.

1881

*Harrington, F. B.
Manton, W. P.
*Swift, W. N.
Titcomb, G. E

1882

Allen, G. W.
Bigelow, E. H.
Buck, H. M.
Miller, G. N.
Morris, J. G.
Tuckerman, F.
Vickery, H. F.
Whitney, H. B.
Woodward, L. F.

1883

Baker, D. E.
Cheever, C. A.
Chisholm, A. S. M.
Cunningham, T. E.
Cutts, H. M.
Devine, W. H.
Foster, C. C.
Galloupe, C. W.
Guiteras, R.
Hewins, P. W.
Hodgdon, A. H.
Johnson, F. M.

1883 (Cont.)

Scofield, C. S.
Trumbull, John
Wetherell, A. B.
Wood, H. A.
Worcester, Alfred

1884

Brainard, J. B.
Conant, W. M.
Daniels, F. H.
*Gates, G. W.
Gifford J. H.
Harrower, David
Jackson, Henry
Kilburn, H. W.

1885

Boyd, S. G.
Brewer, G. E.
Faulkner H. K.
Knowles, W. F.
Louis, Isaac
Lovett R. W.
*Munro, J. C.
Munro, W. L.
Sears, G. G.
Swan, W. D.
*Talbot, Ambrose
Townsend, C. W.

1886

Baldwin, F. W.
Brackett, E. G.
Dodge, W. W.
Dunham, E. K.
Graves, C. B.
Greene, R. W.
Holcombe, C. H.
Howe, O. H.
Jack, E. S.
Mead, G. N. P.
Richards, G. L.
Tuttle, A. H.
Twitchell, E. T.

1887

Chase, H. L.
Clark, J. P.
Cowles, W. N.
Davenport, J. H.
Dunham, Carroll
Fallon, M. F.
Gage, Homer
Jacobs, H. B.
Kaufman, F. J.
Lilienthal, Howard
O'Meara, M. J.
Peters, J. M.
Sears, H. F.
Smith, H. L.

1888

Bryant, W. S.
Chandler, N. F.
*Cushing, E. F.
Eaton, P. J.
Gilman, W. R.
Harrington, T. F.
Pierson, E. L.
Prescott, W. H.
Sargent, G. A.
Scudder, C. L.
Stone, A. K.
Thorndike, Augustus
Thorndike, Paul
Trevino, M. F.
Walker, J. B.

1888 (Cont.)

Wenzlick, Wm.
Yocum, J. R.

1889

Abbott, H. P.
Bonney, S. G.
Chadbourne, A. P.
Clark, L. B.
Day, F. L.
Greenwood, Allen
Halpin, A. J.
Harding, G. F.
Hunting, N. S.
Lancaster, W. B.
Mahoney, S. A.
Morse, C. E.
Peterson, Reuben
Phippen, Hardy
Storer, Malcolm
Thayer, W. S.

1890

Bancroft, G. A.
Bell, R. M.
Carpenter, T. B.
Carroll, F. E.
Churchill, F. S.
Craigin, G. A.
Foote, E. M.
Goldthwait, J. E.
Holden, E. M.
Kenefick, J. A.
*Mumford, J. G.
Wilbur, H. G.
Young, C. D.

1891

Bartol, J. W.
Blake, J. B.
Crockett, E. A.
*Fourtin, E. R. P.
Fuller, D. H.
Huddleston, J. H.
Jelly, A. C.
Kingsley, W. L.
Knight, A. S.
Morse, J. L.
Percy, D. T.
Pratt, C. H.
Swan, W. H.
Taylor, E. W.

1892

Ames, J. L.
August, Albert
Balch, F. G.
Cabot, R. C.
Covell, H. H.
Craig, D. H.
Dane, John
Libby, J. H.
Lund, F. B.
Nichols, J. H.
Porter, C. A.
Towle, H. P.
Washburn, Eliot

1893

Abbott, E. S.
Baker, F. H.
Brewster, G. W. W.
Brown, P. K.
Goodale, J. L.
*Harlow, G. A.
Haskell, H. H.
Jones, C. P.
*Norton, Rupert

1893 (Cont.)

Phelps, J. S.
Piper, Frank
Steese, E. S.
White, C. J.
Yost, F. O.

1894

Allen, W. H.
Bottomley, J. T.
*Cabot, Follen
Chicoine, I. H.
*Cutler, James T.
Darling, E. A.
Fales, A. C.
Hoyt, E. M.
Houghton, H. L.
Keenan, H. J.
Lord, S. A.
Mix, C. L.
Moffit, H. C.
Page, C. G.
Reardon, T. J.
Richardson, M. W.
*Saville, S. C.
Shaw, A. J.
Stevens, C. B.
Swan, C. L.
*Webber, S. E.

1895

Baldwin, H. T.
Capps, J. A.
Chute, A. L.
Codman, E. A.
Cushing, Harvey
Denny, F. P.
Dudley, A. W.
Farnham, J. M. W.
Hall, Herbert J.
Holmes, E. M.
Joslin, E. P.
Marsh, A. W.
Miller, L. C.
Robey, W. H., Jr.
Smithwick, M. P.

1896

Cummin, J. W.
Drohan, J. H.
Ellsworth, S. W.
Frost, Horace B.
Greenough, R. B.
Howland, J. B.
Hubbard, J. C.
Jones, D. F.
Mac Isaac, J. A.
Newell, F. S.
Potter, N. B.
Schwab, S. I.
Spear, W. M.
Thorndike, Wm.
Torrey, J. P.
Washburn, F. A.
Young, E. B.

1897

Badger, G. S. C.
Briggs, C. E.
Broughton, A. N.
Daly, T. J.
Pearce, R. M.
Shea, J. J.

1898

Bailey, W. C.
Balch, A. W.
Beal, H. W.

1898 (Cont.)

Carey, F. H.
Collin, C. O. L.
Cutler, C. N.
Dearborn, S. S.
Donlan, Chas. E.
Donohoe, Geo.
Duckering, W. W.
Fischer, O. E.
Hudnut, P. A.
Hyde, Frederick T.
Kennedy, Harris
King, M. L.
Mackay, E. H.
May, W. R.
Mellus, Edw.
Moore, J. S.
Rose, W. H.
Stack, C. F.
Truesdale, P. E.
Walker, D. H.
Weil, A. I.
Weis, J. D.

1899

Allen, Freeman
*Churchill, Donald
Crane, C. C.
Davis, E. J.
Dix, G. A.
Dodge, A. M.
Greene, D. C. Jr.
Hammond, W. J.
Kepler, C. O.
Merriam, F. H.
Osgood, R. B.
Rose, A.
Tileston, Wilder
Wilkins, Geo. C.

1900

Bergin, S. A.
Brenneman, R. E.
Brown, Percy
Cannon, W. B.
Coolidge, Sumner
Cox, S. F.
Derby, G. S.
Dunn, C. H.
Eastman, A. C.
Emerson, R. L.
Fuller, C. B.
Griffin, W. A.
Hart, J. S.
Knowlton, J. G. W.
Lincoln, Merrick
Lord, F. T.
Lowell, F. L.
Lowell, W. Holbrook
McKibben, W. W.
Matteson, G. A.
Nightingale, J.
Norton, C. W.
Sanford, H. L.
Seelye, Walter C.
*Shepard, L. D.
Small, A. E.
Swift, H. M.
Taylor, J. D.
Wadsworth, R. G.

1901

Barrett, M. F.
Brady, J. F.
Bragg, L. R.
Breed, N. P.
Bremer, J. L.
Brewer, A. D.
Burley, B. T.

1901 (Cont.)

Burnham, J. F.
Chase, A. A.
Chase, H. M.
Chase, W. G.
Cheever, David
Childs, A. H.
Crane, B. T.
Dutton, Richard
Emerson, Kendall
Flagg, Elisha
Hatfield, H. K.
Jackson, H. B.
Leen, T. F.
Locke, E. A.
Mason, N. R.
Mulherin, W. A.
Murphy, F. T.
Sanborn, G. P.
Sise, L. F.
Skarstrom, Wm.
Taylor, F. L.
Thompson, P. H.
Townsend, David
Underhill, S. G.
Walker, W. D.
Wood, N. K.

1902

Adams, C. S.
Barrows, A. A.
Bowditch, H. I.
Bufford, J. H.
Buffum, W. H.
Campbell, F. E.
Craigin, D. B.
De Normandie, R. L.
Ellis, R. H.
Emmons, A. B.
Evans, Albert
Feiss, H. O.
Feldstein, S.
Furrer, A. F.
Goodall, H. W.
Goodridge, F. J.
Hamilton, R. De L.
Hammond, R.
Hathaway, G. S.
Hodges, Fletcher
Hollister, R. R.
Hunt, E. L.
Kent, Bradford
Knowles, R. K. B.
Little, George T.
Mills, L. H.
Neilson, J. L.
Palfrey, F. W.
Prescott, H. D.
Sims, F. R.

1903

Adams, Z. B.
Bail, J. W.
Burrage, T. J.
Clark, G. O.
Cook, P. H.
Emery, W. C.
Fitch, R. R.
Floyd, C.
Gafney, H. D.
Hawes, J. B., 2d
Homans, John
MacLeod, W. P.
Oakman, C. S.
Parker, E. L.
Sargent, W. L.
Smith, H. B.
Stone, M. C.
Sullivan, E. C.

1904

Balboni, G. M.
Barney, J. D.
Bigelow, Edw. B.
Clow, F. E.
Cushing, A. A.
Cusick, L. F.
Clark, G. W.
Drowne, E. L.
Easton, C. D.
Fabyan, M.
Field, H. M.
Finkelstein, H.
Gray, C. P.
Hartwell, J. B.
Hatch, Royal
Hosley, W. A.
Howe, G. P.
Kahn, I. S.
Kidner, F. C.
Linenthal, Harry
Lloyd, H. D.
Marshall, H. K.
Mendelsohn, Louis
Oliver, E. L.
Place, E. H.
Phillips, J. C.
Phippen, W. G.
Robbins, W. B.
Russell, C. B.
Sears, E. P.
Seymour, Malcolm
Stanley, F. G.
Strauss, Sidney

1905

Adams, C. W.
Amsden, G. S.
Beach, S. J.
Blake, Gerald
Burnett, F. L.
Chace, F. A.
Chase, H. A.
Dana, H. W.
Day, H. F.
Dexter, Richard
Eastman, T. J.
Ehrenfried, A.
Fisher, Carl
Hoyt, C. W.
Lee, R. I.
Miller, M. D
Morrill, G. N.
Ordway, Thomas
O'Shea, J. H.
Osgood, Geo.
Overlander, C. L.
Shattuck, G. C.
Sibley, B. E.
Stearns, R. S.
Talbot, F. B.
Whitney, J. L.

1906

Adamian, P. A.
Boothby, W. M.
Boyd, D. H.
Collins, A. N.
Crosbie, A. H.
Frothingham, C., Jr.
Gafforio, P. J.
Green, R. M.
Hamilton, F. A.
Kinnicutt, Roger
Ladd, W. E.
McGrath, B. F.
Mixter, C. G.
Mixter, W. J.
Richardson, E. P.
Risley, E. H.

1906 (Cont.)

Rowley, J. C.
Sawyer, W. A.
Sylvester, P. H.
Van Nuys, F.

1907

Brown, L. T.
Cahill, J. W.
Dailey, M. A.
Day, C. O.
Farnsworth, G. B.
Freese, J. A.
Hendricks, H. V.
Higginbotham, F. A.
Huntington, J. L.
Leake, J. P.
Long, A. D.
McDonald, C. A.
Peabody, F. W.
Pratt, M. R.
Sheahan, G. M.
Smith, R. M.
Spooner, L. H.
Stanwood, F. A.
Swift, W. B.

1908

Adler, H. F.
Edwards, M. R.
Hartshorne, Isaac
Keever, H. F.
Lane, C. G.
Lawrence, C. H.
Morrison, H.
Newburgh, L. H.
Smith, G. G.
Soule, W. L.
Swift, J. B., Jr.
Whittemore, W. S.

1909

Bowditch, Harold
Brigham, F. G.
Dennen, R. W.
Fitz, Reginald
Greeley, H. P.
Macomber, D.
Overlander, J. E.
Parker, Willard S.
Preble, W. E.
Reid, W. E.
Young, E. L., Jr.

1910

Barkan, Hans
Cutler, G. D.
Forbes, Alexander
Gamble, Jas. L.

1911

Austin, Richard S.
Bowers, G. F. H.
Clymer, George
Eustis, R. S.
Forbes, H. S.
Greenbaum, J. V.
Hornor, Albert A.
Jones, Ellis
Means, J. H.
Reynolds, R. L.
Smith, William David
Whidden, Rae W.

1912

Anderson, W. G.
Bell, R. D.
Hackett, L. W.
Lyman, Henry
Minot, Geo. R.
Rackemann, F. M.
Reggio, A. Wm.
Talbot, John E.
Woody, McIvor

1913

Blake, Francis G.
Buffum, W. P., Jr.
Cutler, E. C.
Denny, G. P.
Favill, John
Grinnell, F. B.
Hill, Lewis W.
Robertson, O. H.
Shapira, Albert A.
Strong, Seth L.
Wentworth, E. T.

1914

Binney, G. H.
Cobb, Stanley
Fernandez, M. J.
Gray, Horace
Lehman, E. P.
Levine, Samuel
Lunt, Lawrence K.
Lurier, Israel
Ohler, W. Richard
Richardson, H. B.
Solomon, H. C.

1916

Donation from Class

* Deceased.

ADMITTANCE OF WOMEN TO COLUMBIA MEDICAL SCHOOL.—The Medical School of Columbia University has decided upon the admittance of women in equal standing with men to its medical courses. This arrangement has been made possible by the gift of $50,000 from George W. Breckenridge of San Antonio, Texas. While the question of admitting women to the school has been under advisement for a long time, the decision was hastened by the altered conditions brought about by the war.

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ERNEST GREGORY, *Manager,*
126 Massachusetts Ave., Corner Boylston St., Boston, Massachusetts.

THE FREE DIAGNOSIS OF PATHOLOGICAL MATERIAL.

THE State Department of Health announces in its Monthly Bulletin* the inauguration in Massachusetts of a service for the free diagnosis of pathological material. Such a service is already in successful operation in a number of other states, through the agency of the laboratories of State Universities or the State Departments of Health. In Massachusetts the service has been offered by the Cancer Commission of Harvard University, and the work will be under the supervision of Professor James Homer Wright, pathologist of the Massachusetts General Hospital, whose reputation is such as to secure the highest standard of scientific accuracy. Full details in regard to the shipment of specimens may be obtained from the district health officers of the state, and reports will be sent, in duplicate, to the health officer of the district, as well as to the physician who submits the material for diagnosis.

* Quoted in another column of this issue of the JOURNAL, page 533.

The larger hospitals of the state are, of course, supplied with pathologists and laboratory facilities competent to provide accurate pathological reports upon the tissues removed at operations. Outside of these hospitals, however, and especially in the case of poorer patients who cannot afford the fee for a private pathological examination, this measure of accuracy is too frequently neglected. The institution of a free diagnosis service will bring the advantages of such an examination within the reach of every resident of the state who consults a registered physician.

The most important aspect of this movement is in its application to the surgery of malignant disease, and it is largely in response to the recommendation of the American Society for the Control of Cancer, and of the Committee on Cancer of the Massachusetts Medical Society, that the work is undertaken in Massachusetts. The material submitted for examination will be derived chiefly from operations for the radical cure of carcinoma and sarcoma, and from less extensive operations for the removal of tumors and conditions which are clinically non-malignant, but which are known to possess possibilities of malignant change. While all tissues removed at operation should be subjected to examination, it is expressly stated in the announcement that the employment of these facilities for the diagnosis of small fragments of tissue removed by exploratory incision is not countenanced. The best surgical opinion of the present day absolutely condemns the incision into cancer tissue for the removal of a fragment for later microscopic diagnosis, on the ground that such a procedure is likely to lead to rapid spread of the disease. Only when facilities for immediate frozen-section diagnosis are available, and the radical operation, if it prove necessary, can be completed at one sitting, is such a measure even to be tolerated, and then only when all other resources of diagnosis have been exhausted, and the mutilation of the radical operation is so great as to outweigh the advantages of greater prospect of cure, when the exploratory incision is avoided.

Not a small part of the advantage of such a service will be the general increase in knowledge on the part of the laity, as well as of the medical profession, of the earliest symptoms of malignant disease. We may feel sure that another and an important step has been taken toward the much-to-be-desired control of cancer.

POLLENOSIS.

The reality of the condition known as pollenosis, or hay-fever, has been well established by experimental work in the field of immunization and treatment with pollen extracts. Although hay-fever is caused by the pollen of plant life, it is far from being a disease of the country, but is perhaps just as common in the cities. In all probability the urban community has a greater susceptibility owing to the adverse mode of urban life. The mongrel weeds found so widely distributed along the streets, open lots, backyards, etc., are potent factors in keeping the disease alive in the city. Chicago has recently undertaken a campaign of extermination against the stray weeds of the city, much as the city would undertake a campaign against any public nuisance or menace to health. The extent of the pollen nuisance was demonstrated in the City of Hamburg, by Liefmann, who found 250 pollen grains to the square centimeter. With the increased demand for garden planting there is less excuse for allowing weed plots that might be profitably converted into garden patches for the increase of the food supply.

Although the pollen grain is so widely distributed that the mucous membranes of every individual come in contact with it, only those who are susceptible contract the disease. The susceptibility to pollen infection increases generally with the lowering of the vitality, with nasopharyngeal disease or obstruction, with an increase in the amount of pollen inhaled—and, further, there seems to exist a hereditary sensitiveness to the disease in certain individuals. The spring variety of hay-fever is caused by the Graminaceae family of plants,—the autumn by the Compositae, especially the ragweed. More generally, hay-fever is caused by the pollen of flowers, various grasses, shrubs and even trees.

The disease is characterized clinically by an exudative catarrhal inflammation of the nasal, tracheal, bronchial and conjunctival mucous membranes, together with certain subjective symptoms. Hay-fever is a toxaemia; it is a hypersusceptibility to a foreign protein. The predisposition to this disease is due to an anaphylaxis established for these pollens. The toxin has a selective action upon the nerve cells, particularly upon the autonomous centers, producing an irritability in these centers, which in turn produce congestion in the erectile tissues of the nose, and in the allied mucous membranes, asthmatic symptoms, irritability of the motor areas, nervous excitation, destruction of mucous membrane, etc. When the pollen is brought into contact with the respiratory mucous membrane, either the pollen may be broken down into harmless products, such as proteoses and amino-acids, or the reduction process or cleavage by the mucous membrane may result in toxic proteins. But even the toxic cleavage in susceptible individuals may proceed so slowly that at no time are sufficient toxic proteins released to produce any symptoms, because the mucous membrane can take care of a certain amount of the pollen toxin. But in a suitable case the cleavage takes place rapidly and violently with the release of the specific enzymes and the production of the characteristic symptoms of the disease. Anaphylaxis may be established to a particular pollen or to a family of pollen-producing plants. The study of the specific vaccine treatment of pollenosis has brought to light the fact that the blood of affected patients or of animals used in experimentation contains amboceptors, precipitins and antitoxins.

The only form of treatment which promises any degree of success in pollenosis, is the vaccine treatment with pollen extract. Failures may be ascribed entirely to the fact that the pollen extracts used have not embraced any of the varieties of pollen that caused the anaphyaxis to the disease. The polyvalent pollen extract ordinarily used can no more be expected to reach every variety of pollen infection than polyvalent vaccines against bacterial infections. Usually, however, sensitiveness to the proteins of one pollen produces sensitiveness to the proteins of others of the same family, although not, of course, to the same extent. In practical work a combination of the Graminaceae and Compositae families reaches every indication. As in bacterial antitoxin work, the unit is the standard in immunization and treatment. The unit is an arbitrary factor determined as the quantity of pollen toxin that can be extracted from the thousandth part of a milligram of Phleum pollen. The strength of the pollen extract is determined by the number of such units contained in a cubic centimeter of extract. The extract is diluted before use to the required strength. The measure of resistance to the pollen toxicosis is determined by the number of units necessary to excite conjunctival reaction. As in all immunization work, the first effect is a lowering of the resistance—

the negative phase, followed soon by the increase or positive phase. Reactions are obtained either by instilling the extract into the eye, injecting it between or into the skin, or by the cutaneous route over a scarification. Usually, one-third the number of units necessary to produce the ophthalmic reaction is the dose for the prophylactic treatment, and ranges from 2 to 1,000 units. For therapeutic injections, one-tenth of the number of units giving the reaction are used. Of course, the ophthalmic test cannot be given in the active stage because of the inflammation, and the endermic or cutaneous tests must be used. The increase of the subsequent dosage depends upon subsequent reactions.

MEDICAL NOTES.

WAR MEETING OF THE AMERICAN PUBLIC HEALTH ASSOCIATION.—War and Health will be the central theme at the meeting of the American Public Health Association at Washington, D. C., Oct. 17-20. Some of the speakers on these subjects will be Surgeon-General Gorgas, U.S.A.; Surgeon-General Braisted, U.S.N.; Colonel T. H. Goodwin of the English Army; Assistant Surgeon-General J. W. Trask, U.S. P.H.S.; and representatives of the French and Canadian Sanitary Bureaus.

Each of the seven sections of the Association will have a conference on health problems and opportunities of the war, and a symposium on this subject will be given at one of the general sessions by committees from each section.

Mayors, boards of health, and others interested in sanitary science all over the country are invited to be present or to send representatives to the War Meeting. The Association emphasizes the necessity of obtaining hotel accommodations well in advance, as Washington will be crowded. Non-members are welcome.

The complete program has not yet been published, but will appear in the October number of the *American Journal of Public Health*, to be issued shortly before the meeting. Separate programs may be had by addressing the Acting Secretary, at 126 Massachusetts Avenue, Boston.

Although a large part of the meeting will be devoted to health problems of the war, about half the papers will be of non-military nature.

Following is a partial list of the papers to be presented:

Presidential Address, W. A. Evans, M.D.: Address, Surgeon-General W. C. Gorgas U.S.A.; Address, Surgeon-General W. C. Braisted, U.S.N.; Address, Assistant Surgeon-General, J. W. Trask, U.S.P.H.S.; Address, Col. T. H. Goodwin of the English Army; Address, Franklin H. Martin, M.D., National Council of Defense; Address, Herbert C. Hoover (or a representative), Director U. S. Food Administration; War and Mental Diseases, Major Pearce Bailey, U.S.A.; Tuberculosis and the War, Dr. Hermann M. Biggs, State Health Commissioner, Albany, N. Y.; Report of Committee on Venereal Diseases, Dr. W. F. Snow, Member Advisory Council of Defense; Symposium—Venereal Disease Control in the Army and Navy; Rehabilitation of Injured and Crippled Due to the War, Major Joel E. Goldthwait, M.R.C.; Chairman's Address—Some Public Health Problems of the Present War, Dr. E. C. Levy, Chairman Public Health Administration Section; Chairman's Address—The Service of Health Laboratories in Time of War, Dr. Henry Albert, Chairman Laboratory Section; Sanitation of Barracks and Surrounding Zones—speaker to be announced; Rôle of the Local Health Officer in National Defense—speaker to be announced; Notification of Diseases and Protection of Troops, A. J. Chesley, M.D., Minneapolis, Minn.; General Measures for the Prevention and Control of Industrial Diseases in Time of War, Dr. Alfred Stengel; Need for Sanitary Supervision of Industries in Times of War, Dr. E. R. Hayhurst, Columbus, Ohio; Practical Points in the Safe Handling of T. N. T. and Allied Explosives, Dr. G. W. Hudson; Replacement of Men by Women in War Industries, Josephine Goldmark or Ida Tarbell; Industrial Fatigue, Its Relation to War Industries, Prof. Frederic S. Lee; War Activities of the Bacteriological Laboratories in France and England, Dr. William H. Park, Director of Research Laboratories, New York City; Canadian Laboratories in War Service, Dr. Nasmith, Director of Laboratories, Toronto, Ontario.

POLIOMYELITIS IN NEW YORK STATE.—The town of Colton, St. Lawrence County, New York, has reported eleven cases of poliomyelitis. The schools and churches have been closed to children. No deaths have resulted as yet.

GIFT OF MAYO HOSPITAL TO UNIVERSITY.—The period of affiliation of the Mayo Hospital with the University of Minnesota is at an end and the board of regents of the University have ratified by unanimous vote the permanent agreement making the Mayo Foundation at Rochester the absolute property of the University, to be used perpetually for higher medical education, research and investigation. Securities totaling $1,650,345, representing the fortunes of Drs. William J. and Charles Mayo, were turned over to the university. The expenses of the Foundation will be paid by the Drs. Mayo until a fund of $2,000,000 has accumulated, when the income will be sufficient to maintain the hospital. Under the terms of the agreement, the Foundation cannot be moved to another point in the state until after twenty-eight years.

AMERICAN ASSOCIATION OF CLINICAL RESEARCH.—At the annual meeting of the American Association of Clinical Research held in Boston, the following officers were elected for the coming year: President, Dr. Marshall William McDuffie; first vice-president, Dr. Roger M. Griswold, Kensington, Conn.; second vice-president, Dr. Joseph A. Woitz, Montpelier, O.; secretary-treasurer, Dr. James Krauss, Boston; registrar, Dr. Alonzo J. Shadman, Boston. The next annual meeting of the Association will be held in New York, during October, 1918.

WAR NOTES.

APPOINTMENT OF GASTROENTEROLOGISTS TO BASE HOSPITALS.—The gastroenterological section of the American Medical Association will appoint thirty-two specialists in diseases of the stomach and intestines, who will receive commissions in the Army to serve in base hospitals of the United States.

APPOINTMENT OF BALTIMORE PHYSICIAN.—Dr. Arthur M. Shipley of Baltimore, professor of surgery at the University of Maryland, who had received his commission as chief of the surgical staff of the University of Maryland Base Hospital Unit, has been transferred by the War Department to Camp Meade, where he will become chief surgeon and surgical instructor at the camp.

AWARD OF VICTORIA CROSS.—It is announced that the Victoria Cross has been awarded to Temp. Captain Harold Ackroyd, M.C., for most conspicuous bravery. He was a student at Guy's Hospital and practised at Royston, Herts. Upon entering the service he was attached to the Royal Berks Regiment and was killed in action.

MEDICAL UNIT OF AMERICAN WOMEN.—A mobile hospital unit composed entirely of American women, has been organized, and the French Government has accepted its services. Dr. Caroline Finley is director of the unit, which has the support of the National American Woman Suffrage Association. The work of the service will be principally among the women and children of the devastated districts of France, but in time of need it can be used to care for wounded troops. The unit offered its services to the United States Government, but, because women are not eligible to its medical corps, the offer was refused.

APPOINTMENT.—The appointment of Dr. Arthur True Ellison of Spencer, Mass., as first lieutenant in the United States Medical Reserve Corps, is announced. Dr. Ellison is a graduate of Middlebury College and of Tufts College Dental School.

MEDICAL EXPENSE OF THE WAR.—It has been estimated that the medical upkeep for each soldier of the army will be $25 for the first year. But this total of $25,000,000 for a million men is but a small part of the sum needed to finance the medical department of the army. Surgical dressings will cost $9,750,000; fabrics and textiles, $4,450,000; antiseptics and disinfectants, $3,400,000; motor ambulances, $6,840,000; medical and surgical instruments and appliances, $880,000, besides a wastage, including losses by sea of $16,671,000. Gas masks, trench sprayers and oxygen apparatus will cost $23,000,000. Every man on the front line will be provided with two masks and trained to put them on in six seconds. The standard type of motor ambulance adopted will cost about $2000 and will be heavier than the type previously used. It has been tried out on the border and in Mexico and has proved its satisfaction. The medical department has not been relieved to any great extent by the contributions of various organizations. The department has taken over ten base hospitals, but only four had equipment and that was valued at only $25,000 for each unit. Supplies are far short of what will be needed to equip the enlarged regular army alone. In order that they may be hastened, Surgeon-General Gorgas has been authorized to incur a deficiency for immediate preparatory measures to the amount of $29,000,000 over and above funds now in hand. Under that authority orders have been given to medical purchasing officers to make contracts and to place orders for large quantities of supplies, the exact cost of which it is impossible to estimate at the present time.

RED CROSS SCHOLARSHIPS.—The town and county nursing service of the American Red Cross has received donations of money sufficient to establish a number of scholarships for the purpose of training women in public health nursing. The scholarships, of $250 each, cover an eight months' course at Columbia University, Simmons College, Western Reserve University, University of Cincinnati, and the School of Civics and Philanthropy, Chicago.

AMERICANS IN CHARGE OF BRITISH HOSPITALS.—It is reported that six British hospitals will be put in charge of American doctors. They are at Manchester, Salford, Liverpool, Leeds, Birmingham and Cardiff, and at the first-named place eleven American doctors have been detailed. The British physicians thus released will return to civil practice. The sending of large numbers of physicians to the front has resulted in suffering among the civil population for the need of adequate medical attendance, and it is for this reason that American doctors have been assigned to take charge of the hospitals.

ASSUMPTION OF TUBERCULOSIS WORK.—The trustees of the "Tuberculeux de la Guerre" have transferred their activities to the American

Red Cross. Major Grayson P. Murphy, head of the American Red Cross Commission to Europe, received, on behalf of the Red Cross, the property of the association and assumed control of the work. The property consisted of four sanatoria, about 1,500,000 francs, several automobiles and a large equipment of blankets, food and other materials for the use of patients suffering from tuberculosis. Major Murphy stated, in acceptance, that the great work for the control of tuberculosis in France would be carried on in conjunction with the plans of the Red Cross and the Rockefeller Foundation.

HONORS FOR AMERICAN DOCTORS AND NURSES.—The French Hospital of New York, which has been operating a hospital in Yonne, France, since 1916, was honored recently by Justin Godart, under-secretary for the army medical service. Drs. John Irwin and Thomas Savage, who have served since the hospital was started, were presented with medals of public instruction. Other medals were given to nine of the nurses. The hospital is equipped with 250 beds, and is supported entirely by private funds. Mrs. William Fitzgerald of New York assumed its expense at first, but later it was taken over by Henry Walker of Baltimore.

WAR RELIEF FUNDS.—On October 6 the totals of the principal New England war relief funds reached the following amounts:

French Wounded Fund	$265,031.02
Armenian Fund	235,269.06
Surgical Dressings Fund	134,819.06
Permanent Blind Fund	123,184.03
British Imperial Fund	109,911.00
Polish Fund	87,308.59
Italian Fund	44,543.72

BOSTON AND MASSACHUSETTS.

WEEK'S DEATH RATE IN BOSTON.—During the week ending Sept. 29, 1917, the number of deaths reported was 230, against 238 last year, with a rate of 15.53, against 15.6 last year. There were 65 deaths under one year of age, against 52 last year.

The number of cases of principal reportable diseases were: diphtheria, 79; scarlet fever, 16; measles, 15; whooping cough, 24; typhoid fever, 9; tuberculosis, 67.

Included in the above were the following cases of non-residents: diphtheria, 15; scarlet fever, 7; measles, 1; whooping cough, 2; tuberculosis, 5.

Total deaths from these diseases were: diphtheria, 3; typhoid fever, 1; tuberculosis, 15.

Included in the above were the following non-residents: diphtheria, 1; typhoid fever, 1; tuberculosis, 2.

BABY HYGIENE ASSOCIATION.—During the month of August the Baby Hygiene Association cared for the largest number of babies in its history,—2682. This is 500 more than were cared for a year ago. A survey has been made of the City of Boston in the interests of child conservation which shows that added stations are needed for the Jamaica Plain and Upham's Corner districts. As soon as funds can be raised, these sections of the city will be provided with stations. The Lincoln House has offered the services of its dietitian to the Association to supervise a clinic for children between the ages of one and five years. This clinic will be opened October 1, and will care for the child up to school age,—a period which has had no medical or nursing supervision.

SUSPENSION OF PAY CLINIC.—The Pay Consultation Clinic, which has been in operation at the Massachusetts General Hospital for some while, has been suspended for the time being. The war has caused such a shortage in the staff of the hospital that it has been found impossible to continue this Clinic longer. It is the intention of the Hospital to re-open the Clinic when conditions are more favorable.

HOSPITAL BEQUESTS.—By the will of the late Mrs. Augusta E. Corbin of Boston, the Massachusetts Homeopathic Hospital is the recipient of a gift of $100,000, the income to be used for general purposes, and the New England Deaconess Association is to receive the sum of $100,000 to erect and equip a building or extend buildings for hospital purposes, and the income of an additional $100,000 to be used for hospital work.

GRADUATION OF DENTAL HYGIENISTS.—The Forsyth Dental Infirmary has graduated a class of thirteen young women trained as dental hygienists. The class was established at the Forsyth Infirmary on October 2, 1916, in conformity with the Massachusetts registration law of 1915, which permits the employment of dental hygienists in public schools and institutions. Pupils are trained in the theory of dental practice, the sterilization and care of instruments, anesthetics and their exhibition, and the care of the patient before, during and after their administration.

NEW QUARTERS FOR STATE HEALTH DEPARTMENT.—The State Department of Health has been moved from the third floor of the State House to the fifth floor of the west wing. The water and sewage laboratories will occupy adjoining rooms and the food and drug laboratories are on the same floor. The only division which will not be located on the fifth floor is the engineering division, which has been on the first floor for many years.

TUBERCULOSIS LECTURES AT FRAMINGHAM.—The Community of Health Station at Framingham has arranged with the Framingham Medical Club to give, during the winter, a course of lectures on tuberculosis. These lectures will

constitute a thorough post-graduate course in this subject, and will bring to Framingham men who have become well known in tuberculosis work throughout the country. The first lecture was given on September 20 by Dr. William Smith, of the Harvard Medical School, who talked on "Differential Diagnosis in Tuberculosis."

The health station has recently been visited by Surgeon Major E. Rist of Paris, who is in America with the French scientific mission. After calling at the health station, Major Rist made a tour of Framingham, and will carry with him, for possible use among the French civilian population, as well as among French military forces, the methods employed here in the detection and treatment of tuberculosis and other preventable diseases.

Another visitor to the Health Station was Dr. H. A. Patterson of the National Association for the Study and Prevention of Tuberculosis, who is in coöperation with Washington authorities in the campaign for the detection of tuberculosis in the United States Army. Under his direction, arrangements have been completed with the military authorities for a thorough examination by tuberculosis experts of all men assigned to the different cantonments.

WOMEN AT HARVARD MEDICAL SCHOOL.—Plans for the admittance of women to Harvard Medical School have been abandoned, for the present at least. The reason lies in the small number of women who made application, and the failure of all but one of these applicants to meet the necessary requirements.

Miscellany.

FREE DIAGNOSIS OF PATHOLOGICAL MATERIAL.

THE State Department of Health takes pleasure in making the following announcement, made possible through the kindly offer of the Cancer Commission of Harvard University.

On and after October 1, 1917, with the cooperation of the Cancer Commission of Harvard University, the State Department of Health of Massachusetts will be able to offer to the registered physicians of the State the opportunity for the free diagnosis of pathological material removed at operation. This service is already provided in a number of states by the laboratories of state universities or by the departments of public health. It is believed that a material decrease in the number of advanced and inoperable cases of cancer, and a considerable increase in the accuracy of diagnosis of cancer and other tumors, will result from the institution of this service.

Specimens submitted for examination will be received at the laboratory of the Cancer Commission, at the Medical School of Harvard University, Longwood Avenue, Boston. This service will be under the direction of Prof. James Homer Wright, M.D., S.D., pathologist of the Massachusetts General Hospital. Reports will be forwarded by mail or telegram to the attending physician.

The practice of cutting through sound tissue into cancer tissue in order to remove a fragment for subsequent microscopic examination is generally condemned, on the ground that such a procedure leads to a rapid spread of the disease and usually deprives the patient of his one chance of cure by radical operation. When doubt exists as to the nature of a tumor which is deeply placed, after all other resources of diagnosis, such as the x-ray, the Wassermann reaction, the blood examination, and competent consultation, have been exhausted, the resources of some hospital or institution equipped for immediate frozen section diagnosis should be sought, and the operation should be completed under one anesthesia. Thus lesions which are suspected to be malignant may be divided arbitrarily into two groups, as follows:

Group 1. Lesions which are superficial or ulcerated, as those of the external skin, the mouth, tongue, palate, larynx, esophagus, rectum, bladder and cervix of the uterus. In such cases the whole lesion should be removed in preference to cutting into the suspected tissue. Where total removal would cause so much mutilation as to be unjustified on a doubtful diagnosis, facilities for a frozen diagnosis should be obtained in order that the radical operation may be completed immediately if malignant disease is discovered. The cautery should be used in these cases to seal the lymphatics while waiting for the report of the pathologist. This caution applies also to suspected carcinoma of the body of the uterus, and immediate frozen section diagnosis of the material obtained with the curette should be made available, and the radical operation, if necessary, performed under one anesthesia. In but few cases is the exploratory removal of a fragment of tissue to be sent for a subsequent pathological examination justified. When no other recourse is open to the physician, however, the specimen should be removed with the cautery, or the cautery should be used immediately to seal the tissue spaces.

Group 2. Lesions which are deeply placed, and covered by sound tissue, as those of the breast, the salivary glands, thyroid, stomach, intestine, gall bladder, kidney, ovary, testicle and tumors of the subcutaneous tissues, lymph nodes, bones, fasciae and muscles. In such cases the exploratory incision into the suspected tumor tissue is *never* justified unless the frozen section diagnosis is available, and even then the radical operation, without exploratory incision is usually safest for the patient. Especially is this true in the case of tumors of the breast.

Benign tumors and diseases which are recognized as particularly liable to malignant devel-

opment should be removed entire, and the tissues submitted for confirmation of the diagnosis by pathological examination. Among these conditions may be included the benign tumors and chronic diseases of the breast, ovarian cysts, papillomata of the rectum and anus, polyps of the uterus, rectum and larynx, and pigmented moles, naevi, warts, fissures and chronic ulcerations of the external skin, especially those which show a tendency to ulceration, bleeding or gradual increase in size.

Containers and full directions for the preservation and shipment of specimens for diagnosis may be obtained from the State District Health Officer. These officers are stationed as follows:

Dr. Adam S. MacKnight, 355 North Main Street, Fall River.

Dr. Merrill E. Champion, 50 Phillips Street, Wollaston.

Dr. Arthur A. Brown, Willey House, Swampscott.

Dr. Charles E. Simpson, 100 Holyrood Avenue, Lowell.

Dr. George T. O'Donnell, 768 Main Street, Waltham.

Dr. Francis A. Finnegan, 14 Longwood Avenue, Fitchburg.

Dr. John S. Hitchcock, 160 Main Street, Northampton.

Dr. Howard A. Streeter, 740 Dalton Avenue, Pittsfield.

A card will be provided which must be filled out by physicians desiring to avail themselves of this service. Reports will go in duplicate to the physician and to the health officer of the district, and any questions raised by the pathological report will be discussed by letter or by personal interview with the Director of the State Diagnosis Service during his office hours, Monday, Wednesday and Friday, 4 to 5 p.m., Cancer Commission, Medical School of Harvard University, Building E, Longwood Avenue, Boston.

CANCER DECALOGUE PREPARED BY THE STANDING COMMITTEE ON THE CONTROL OF CANCER OF THE MASSACHUSETTS MEDICAL SOCIETY.

1. The classical signs of cancer are the signs of its incurable stages. Do not wait for the classical signs.

2. Early cancer causes no pain. Its symptoms are not distinctive, but should arouse suspicion. Confirm or overthrow this suspicion immediately by a thorough examination and, if necessary, by operation. The advice, "Do not trouble that lump unless it troubles you," has cost countless lives.

3. There is no sharp line between the benign and the malignant. Many benign new growths become malignant and should, therefore, be removed without delay. All specimens should be examined microscopically to confirm the clinical diagnosis.

4. Precancerous stage. Chronic irritation is a source of cancer. The site and the cause of any chronic irritation should be removed. All erosions, ulcerations, and indurations of a chronic character should be *excised*. They are likely to become cancer.

5. Early cancer is usually curable by radical operation. The early operation is the effective one. Do not perform less radical operations on favorable cases than you do on unfavorable ones. The chances for a permanent cure are proportionate to the extent of the first operation. Make wide dissections; incision into cancer tissue in the wound defeats the object of the operation and leads to certain local recurrence.

6. Late cancer is incurable, though not always unrelievable. Radium, x-rays, ligation, cautery, or palliative operations may change distress to comfort and may even prolong life.

7. Cancer of the breast. All chronic lumps in the breast should be removed without delay. Benign tumors can be removed without mutilation. Examine all specimens microscopically. An *immediate* microscopical examination is desirable since, if positive, it permits a radical operation at the same sitting. A radical operation performed ten days after an exploration is almost never successful in curing cancer of the breast.

8. Cancer of the uterus. Any irregular flowing demands thorough investigation. Offensive or even very slight serous flows are especially suspicious. Curette and examine microscopically. Amputate all eroded cervices which do not yield promptly to treatment. Do not wait for a positive diagnosis.

9. Cancer of the digestive system is difficult of early diagnosis, and therefore unfavorable in prognosis. All persistent and recurring indigestions (more especially if attended by change of color and loss of weight) and any bleeding or offensive discharges demand prompt and thorough investigation. Do not wait for a positive diagnosis.

10. Cancer of the skin. Any warts, moles, or birthmarks which enlarge, change color, or become irritated, should be removed promptly. They are likely to become cancer. Do not wait for a positive diagnosis.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS, FOR AUGUST, 1917.

GENERAL PREVALENCE.

THERE were 3,176 cases of communicable diseases reported during August, a marked decrease as compared with the total of 4,600 cases reported for July. In August, 1916, 3,172 cases were reported.

Diphtheria, pulmonary and non-pulmonary forms of tuberculosis show an increase in the State, compared with August, 1916.

Customarily, the incidence of diphtheria should decrease from July to August, but this year it remains practically stationary, 597 cases being reported in July and 592 in August.

Anthrax.—Two cases of anthrax were reported. One, confirmed bacteriologically, was a freight handler who had presumably been handling raw hides from South America. The other case was an embosser of finished leather. Laboratory results were negative, but the use of antiseptic solutions may have interfered with the tests. Smears and cultures of suspected anthrax cases should be taken at once, before treating, to insure finding the anthrax bacilli.

Smallpox.—One case of smallpox in Ware was a travelling salesman who had been in Connecticut and Worcester, Massachusetts, a short time before the onset. The patient was vaccinated in 1902, and the case was very mild. There were no cases reported from Worcester in August.

Cerebrospinal Meningitis.—There were 19 cases of this disease reported, a larger number than usual for August. The cases were all scattered, no locality reporting more than 2 cases except Boston, where 4 were reported.

Poliomyelitis.—The danger of an epidemic of this disease is now practically over, as only 39 cases were reported during August, an increase of 1 case over July. The only unusual prevalence of the disease was in Haverhill where 20 cases were reported.

Diseases on the Premises of Milk Handlers.—During the month, 2 cases of typhoid fever, 5 cases of septic sore throat and 2 cases of diphtheria occurred in families of milk handlers.

EPIDEMICS AND OUTBREAKS.

Dysentery.—During July and August 75 cases of dysentery occurred at the Grafton State Hospital, 45 being in the Worcester Department with 3 deaths, and 30 in the Grafton Colony, the cases being confined to two buildings.

The cases in the Worcester Department were confined to the inmates, but in the Grafton Colony two nurses and two staff physicians had the disease.

The disease was characterized with a sudden onset, fever, abdominal pain, frequent copious watery stools occasionally accompanied with blood and mucus, heavy white coated tongue. The duration of the attack averaged four days.

Laboratory examinations failed to disclose any amebae or known strains of Bacilli Dysenteriae. No milk or food handlers were sick, and the source of the outbreak was not determined.

Typhoid Fever.—An epidemic of 54 cases occurred in Gardner due to a carrier on a milk farm.

Streptococcic Sore Throat.—An epidemic of streptococcic sore throat occurred during the first week of August in Natick, Wellesley and Dover. There were 119 known cases of the disease and two deaths, due to streptococcic septicemia.

Attention was first called to the presence of an epidemic by a patient developing a peritonitis and the finding of hemolytic streptococci in the peritoneal fluid.

An immediate investigation showed the majority of the cases to be on the route of one distributor who was supplied by three farms, the C., H. and M. farms.

The investigation showed the presence of hemolytic streptococci in the throat of two milk handlers on the C. farm, and in the throat of one of the milk distributor's family. Streptococci were also found in the throat of a milk handler working for the distributor, but the hemolytic character of the organisms was not determined.

Also one cow on the C. farm was found to be discharging large numbers of non-hemolytic streptococci.

The epidemic was stopped by the isolation of all known cases on the farms, sterilization of milk utensils, pasteurization of milk and the elimination of the cow shedding streptococci from the herd.

RARE DISEASES.

Anterior Poliomyelitis was reported from Acton 1, Boston 1, Brockton 2, Fall River 1, Haverhill 20, Hingham 1, Lowell 1, Lynn 2, Marlboro 1, Natick 2, Northampton 1, Saugus 1, Stoneham 2, Winchendon 2, and Worcester 1.

Cerebrospinal Meningitis was reported from Barnstable 1, Boston 5, Fall River 1, Gloucester 2, Lawrence 1, Lowell 2, Lynn 1, New Bedford 2, Peabody 1, Pittsfield 1, Somerville 1, and Springfield 1.

Dog-bite was reported from Chelsea 1, Lowell 1, and Waltham 1.

Dysentery was reported from Boston 39, Fall River 2, Greenfield 5, Newton 1, and Peabody 1.

Glanders was reported from Tewksbury 1.

Malaria was reported from Boston 2, Brookline 1, Cambridge 1, Danvers 1, Dedham 4, Foxboro 1, Medfield 1, Milford 1, and Northampton 1.

Pellagra was reported from Boston 1, Danvers 1, Peabody 1, and Tewksbury 1.

Septic Sore Throat was reported from Boston 6, Dover 1, Natick 26, Newton 1, and Wellesley 2.

Smallpox was reported from Ware 1.

Tetanus was reported from Brockton 1, Dennis 1, New Bedford 1, Peabody 1, Springfield 2, Westfield 1, and Winchester 1.

Trachoma was reported from Boston 5, Brockton 1, Lynn 1, Peabody 1, and Worcester 2.

Trichinosis was reported from Boston 1.

Correspondence.

A LAY DESCRIPTION OF DIABETES.

Prouts Neck, Me., Sept. 27, 1917.

Mr. Editor:

The following is from Bazán's "The Swan of Villamorta," and interestingly describes the sensations of Don Victoriano Andres de la Comba, a Spanish statesman.

"His words were a monologue, the spoken utterance of the gloomy thoughts which Don Victoriano—thanks to heroic efforts!—had hitherto been able to conceal in his breast.

"The strange malady from which he suffered gave rise to horrible nightmares. He dreamed he was turning into a loaf of sugar and that his intellect, his blood, his life were flowing away from him through a deep, deep channel, converted into syrup. In his waking moments, his mind refused to accept, as one refuses to accept a humiliation, so strange a malady. Sanchez del Abrojo must be mistaken; his was some functional, transitory disorder, an ordinary ailment, the result of his sedentary life, and Tropiezo's old-fashioned remedies would, perhaps, after all, prove more efficacious than those of science.

"And if they did not? The statesman felt a cold chill run through him that made his hair stand on end and constricted his heart. To die when he was scarcely past forty, with his mental powers unimpaired, with so many things begun, so many accomplished! And, no doubt, this consuming thirst, this insatiable voracity, this debilitating sensation of melting away, of fusion, of dissolving, were all fatal symptoms."

It is seldom that one finds in fiction so vivid, and at the same time so accurate, description of the symptoms of disease as this thumb-nail sketch of diabetes by Bazán.

Yours truly,

WILLIAM PEARCE COUES.

NOTICE.

CENSORS' EXAMINATION.

The Censors of the Suffolk District Medical Society will meet to examine candidates for admission to the Massachusetts Medical Society at 8 The Fenway, on Thursday, Nov. 1, 1917, at 4 P.M.

Candidates, who must be residents of Suffolk District or non-residents of Massachusetts, should make personal application to the Secretary, and present their medical diplomas, at least three days before the examination.

For further particulars, apply from 2 to 4 P.M. to

GEORGE GILBERT SMITH, *Secretary.*

99 Commonwealth Avenue.

RECENT DEATHS.

JOHN BERNARD DONNELLY, M.D., a Fellow of the Massachusetts Medical Society, died at his home in West Gardner, August 2, 1917, aged 50 years. He was a graduate of Holy Cross College in 1890, and of the Harvard Medical School in 1893.

MOSES GREELEY PARKER, M.D., a retired Fellow of the Massachusetts Medical Society, died in Lowell, October 1, 1917, aged 74 years. He was a native of Dracut, the son of Theodore and Hannah Greeley Parker, and graduated from Harvard Medical School in 1864, having been assistant surgeon to the Fifty-Seventh Massachusetts Infantry during the Civil War. He made a specialty of diseases of the eye and ear, was visiting physician at St. John's Hospital for 30 years, and a trustee of the Lowell General Hospital up to the time of his death. He was always much interested in the telephone business and in patriotic societies.

HARRY VERNON WEAVER, M.D., died at his home in New Bedford, September 21, 1917, aged 47 years. He was a graduate of Boston University School of Medicine in 1893, practised ophthalmology, otology, laryngology and rhinology and was a Fellow of the Massachusetts Medical Society and also of the Rhode Island Medical Society.

MARRIAGE.

DR. FREDERIC O. WEST of Woburn Mass, was married recently to Miss Gretchen R. Van Tassel, also of Woburn. Dr. West is a graduate of Harvard Medical School, class of 1908.

SOCIETY NOTICES.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY.—A regular meeting of the Middlesex East District Medical Society will be held at the home of the president, Dr. H. A. Gale, Winchester, on Wednesday, Oct. 17, 1917, at 8 P.M. The general topic of the meeting will be Obstetrics.

H. B. JACKSON, M.D., *Acting Secretary.*

WORCESTER DISTRICT MEDICAL SOCIETY.—A regular meeting of the Society was held Wednesday, October 10, 1917, at 4.15 p.m. in G. A. R. Hall, 55 Pearl Street, Worcester.

Program.

1. Principles and Problems of So-called "Health Insurance," Dr. Walter P. Bowers, of Clinton.
2. Discussion.

The Censors meet for the examination of candidates for Fellowship at 4.00 p.m., Thursday, Nov. 1, 1917, in the reference department of Worcester Public Library. Applicants should secure blanks from the Secretary, fill out and file with him on or before Oct. 25. It is also necessary to present diplomas for verification on or before the examination day. The examination is oral.

Revised list of physicians in Military Service of the United States or the Allies.

U. S. ARMY: Dr. Joseph O'Connor, Worcester; Dr. Howard Beal, Worcester; Dr. Edw. B. Bigelow, Worcester; Dr. Frank W. George, Worcester; Dr. Roger Kinnicutt, Worcester; Dr. E. B. Simmons, Worcester; Dr. William E. Denning, Worcester; Dr. Willard Lemaire, Worcester; Dr. James J. Goodwin, Clinton; Dr. Chester C. Beckley, Lancaster; Dr. Roger Schofield, Worcester; Dr. Merrick Lincoln, Worcester; Dr. George Lincoln, Worcester; Dr. E. S. Phelan, North Brookfield; Dr. R. S. Newton, Westboro; Dr. William J. Fay, Worcester; Dr. A. K. Yoosuf, Worcester; Dr. James V. May, Worcester; Dr. Gilbert Haigh, Worcester; Dr. Samuel C. Gwynne, Worcester; Dr. William N. Stapleton, Worcester; Dr. Harry P. Cahill, Worcester; Dr. Israel Lurier, Worcester.

U. S. NAVY: Dr. Louis Johnson, Worcester; Dr. Thomas Courtney, Worcester; Dr. Joseph L. Lannois, Northboro; Dr. Winthrop Adams, Worcester.

ENGLISH ARMY (Harvard Unit): Dr. Kendall Emerson, Worcester; Dr. Oliver Stansfield, Worcester; Dr. Stanley Bridges, Worcester; Dr. George Watt, Worcester.

ENGLISH HOSPITALS: Dr. Albert O. Raymond, Worcester; Dr. William H. MacKay, Worcester.

The secretary is in receipt of a letter from Dr. Edw. H. Mackay, stating that the inclusion of his name in the Army list last month was an error.

The Medical War Committee invites information to supplement or correct this list. Doctors joining the Colors should notify the committee and indicate their wishes with regard to sharing the fees derived from their patients. ERNEST L. HUNT, *Secretary.*